ANNALS *of* SURGERY

VOL. LXXV APRIL, 1922 No. 4

DRAINAGE*

BY JOSEPH A. BLAKE, M.D.

OF NEW YORK, N. Y.

As far as the drainage of wounds of the muscle planes is concerned the practice, as generally carried out, seems to be satisfactory and does not call for much improvement. During the war, the importance of dependent drainage was emphasized by our experience with gunshot injuries, but we also found that drainage was not necessary when there was no dead or necrotic tissue in the wound. It is well to bear this last fact in mind, for it is also true in infections of the body cavities and viscera. I am now speaking of drainage in the sense of employing tubes or other materials to produce it, or rather continue it; for we should make a distinction between an incision used merely to evacuate a cavity of the products of inflammation and perhaps allow these products to escape for a short time, and drainage continued for a longer period by means of drains. I shall use the term evacuation for the former and reserve drainage for the latter procedure. An illustration of the needlessness and, as I shall show later, the actual harm of drainage when there is no necrotic tissue remaining, occurs in the treatment of peritonitis. I called attention to this in 1903–04 † and at that time showed a marked difference in mortality in favor of non-drainage between drained and undrained cases of bad diffuse peritonitis. It is only fair to state that the high mortality followed introduction of drains through multiple incisions and also much handling of the viscera; yet subsequent experience demonstrated that the drains themselves by pressure and foreign body irritation, or presence, or whatever you may call it, kept up a peritonitis which otherwise would have subsided. At that time I made the rule that drains into the peritoneal cavity were unnecessary when, after elimination of the cause of a peritonitis, there was no great difference in the appearance of one part of the peritoneum from another. Thus, in an ordinary diffuse peritonitis no matter how bad the peritonitis, drainage was unnecessary, but in a localized peritonitis, as, for example, an abscess, drains should be used.

The same principle of non-drainage in diffuse infections without local

* Read at a joint meeting of the New York Surgical Society and the Philadelphia Academy of Surgery, November 7, 1921.

† Treatment of the Peritoneum in Diffuse Peritonitis, ANNALS OF SURGERY, August, 1903. The Treatment of Diffuse Peritonitis, New York Medical Journal and Philadelphia Medical Journal, November, 1904.

necrosis was found to hold true during the war in the treatment of infections of the joints. Early in the war I found that drains traversing the joints were fatal to the joint, and my results were greatly improved by using short drains which only entered the joint. Later on we all were struck with the remarkable results obtained by Wilms, who did not use drains at all, but after opening them relied on active motions of the joints for the prevention of accumulation.

In the treatment of empyema we have not as yet been able to demonstrate in so forceful a manner that drainage tubes are detrimental, yet I believe, if we could only insure some other method of keeping the wound in the thoracic wall patent, that much better results could be obtained than by the use of tubes. The nearest I have been able to get to this ideal is by the use of very short tubes; tubes just long enough to keep the pleural cavity empty. These tubes may be removed usually in four or five days, namely, as soon as the drainage tract is established; and in most cases the cavity is closed and the condition cured in ten to twelve days. On the other hand, if tubes traverse the cavity and are left in for longer periods, no matter whether they are slender Carrel tubes or tubes as big as one's thumb, they establish the infection which often becomes mixed in character and cause a more or less chronic sinus.

As I say, it would be better to leave tubes out if we could; but, in order to drain efficiently, the opening should be made just at the upper line of the costo-phrenic sinus, and if continued drainage is needed something must be introduced to prevent the diaphragm closing the opening. In some cases of empyema in which resistance to the infection was obviously active and in which there was extensive fibrinous exudate I have cleaned out the latter by hand through a large incision, and dispensed with drains and obtained immediate resolution.

There is no doubt but that evacuation of the products of infection by simple incision will suffice in many cases of empyema. It only remains to be able to select or rather recognize such cases. It is difficult to lay down rules by which such cases may be distinguished, but I believe it is safe to dispense with tubes if after evacuation no visible foreign material such as adherent fibrin remains. In such cases the secretions forming in the pleura will be serous in character and will escape through the incision, which should not be entirely closed by suture. In two or three days there will be no secretion and no drainage unless foreign materials such as drains are left in the chest. In other words, repair in the pleural cavity does not differ essentially from repair in a joint or the peritoneal cavity.

There is another class of drainage cases in which the question again arises as to whether the drainage we customarily employ is not detrimental in a similar way. I refer to drainage of the common bile duct in cases of cholangitis and more particularly when drainage is to be employed temporarily as is the case in the great majority. The usual manner in which drainage is accomplished is by introducing a tube into the duct. The tube may pass upward into the hepatic duct, or it may be a T tube, one limb of the T lying

in the hepaticus and one in the choledochus. The question is, should we, in view of our knowledge of the harmful effects of drainage tubes in prolonging and establishing infection, introduce tubes into the duct? Would it not be better to simply drain to the opening we have left in the duct? Closing of the opening in the duct thus dispensing with drainage has already been recommended and practiced; but when one has traumatized the duct, particularly if in the region of the papilla, or when pus is flowing from it, one hesitates to close it. An objection to draining only to the opening in the duct is that the bile flows into the peritoneal cavity before entering the drainage tube. This does not seem to matter, for we find it escapes around the tube even when the tube is introduced into the duct. When I have drained to the opening in the duct and not into the duct, it is my impression that the discharge has cleared more quickly with less suppuration and has ceased in a few days instead of often as many weeks.

There is another class of cases in which drainage of the viscus is not practiced frequently enough. I refer more particularly to cases of advanced peritonitis with paralytic ileus. How often the peritoneal cavity has been drained when unnecessary and the patient has died of ileus, often caused, it is true, by the drains in the peritoneum, but more often by toxic paralysis of the muscularis or a combination of paralysis and tubes. Most of us have saved patients by a secondary ileostomy. My plea is that it should more often be a primary operation. Not that it should be done in every case, but that it should be done more often. It is very easy. A soft catheter stitched with catgut to the margins of a small opening in the lower part of the ileum and then the wall inverted with a couple of purse-strings so that the stoma will close when the catheter is withdrawn is all that is necessary. The catheter may be withdrawn with safety after two or more days when the catgut stitch has loosened. There is no need of a Paul's tube or any other such formality. I have brought the catheter out of a buttonhole incision in the linea alba when I wished to close the operation wound completely.

There is still another type of drainage to which I shall apply the term *precautionary;* namely, drainage, which is used to prevent accumulation of wound secretions, or infection in case of visceral leaks. I have little or nothing to say of precautionary drainage of ordinary wounds to prevent accumulation of serum or blood, for I believe that the rules for this are sufficiently definite. Of course every surgeon of experience establishes his own, although the question is often debatable as to whether one should drain or not. As a protection against peritonitis caused by leaks there are two operations in which surgeons differ and in regard to which there is a grand opportunity for discussion. These two operations are cholecystectomy and resections of the colon. I believe a drain does no harm in these cases and will save lives. Gauze should not be used. A slip of rubber dam is sufficient. All that is necessary is a lead along which discharges may escape.

If retroperitoneal spaces are opened, and particularly if large as in retroperitoneal ureterotomy, drainage must be employed.

Before closing I must say a word about the materials used to produce drainage. In the first place, gauze is commonly employed, but never should be for drainage alone. Gauze is extremely useful as a packing in certain classes of wounds but is not a good drain and may be exceedingly injurious. In the first place gauze is an effective filter and consequently while the serous secretions may escape through it, it acts as a dam to the solid necrotic portions which form the food upon which bacteria grow. Gauze has been a woeful cause of death when used in gunshot wounds and in the peritoneal cavity. It has plugged up the secretions in one and acted as an irritant and cause of obstruction in the other. The indications for its use are, first, as a pressure hæmostat, and, second, to prevent the soft parts falling into cavities during the early stages of repair such as are formed in excision of joints or other operations upon bones. Except when used as a hæmostat it should be separated from the wound surfaces by rubber or other non-adhering material.

Rubber tubing is the material most commonly used for drainage, but is more often used than is necessary. The true indication is when large quantities of material, and particularly solid material, are to be evacuated. Thus we use tubing with reason when we drain the urinary bladder, the gall-bladder or ducts, or the intestines, and also when we drain wounds in which there is solid necrotic material which has to be evacuated by irrigating through the tubes. For these latter cases, the tubes should be large, for otherwise they may become blocked. When large it is very important for them to be soft as otherwise they may be dangerous through causing pressure necrosis. I shall never forget the sensational report by one of our members of a case of ligation of both iliac arteries because of hemorrhage produced by the pressure of drainage tubes used after a double ureterotomy for calculi. If solid matter does not have to be evacuated there is much to be said in favor of a number of small tubes such as the Carrel tubes, for they are not so likely to cause disagreeable pressure effects and they permit the use of irrigations. Also as the holes in tubes are usually blocked by the soft tissues, Chaput is probably correct in his belief that drainage takes place alongside of and about rather than through tubes. However, even if we agree with him thus far, we should not necessarily go to the point of believing that a bunch of silkworm gut is better for all conditions than a tube because there is more superficies to the many strands of gut than to the single tube. For precautionary drainage silkworm gut and folded rubber dam are excellent, for they do not produce pressure necrosis, they efficiently drain off fluid secretions and also form a sufficient lead along which larger drains may be inserted if developments demand them.

WAR INJURIES COMING TO SUBSEQUENT OPERATION

By Walter M. Jones, M.D.
of St. Louis, Mo.
SURGEON IN THE U. S. PUBLIC HEALTH SERVICE

It is not our intention in this report to review those post-bellum conditions operated in the last two years, as that would involve the perusal of more than 1800 operations. We have been able to improve many injury cases by means other than operation. Many had attained the maximum result before arriving at this hospital, the majority showing most excellent results, no doubt far surpassing the most sanguine expectations of the original operator, while in other cases the results must have fallen short of his expectations; especially is this true of nerve sutures, amputations and bone injuries.

It is not our intention to criticize war surgery, especially when we consider the circumstances under which most of the work was done; indeed the cases are few where we feel that the result obtained was due primarily to a lack of surgical knowledge.

The surgeon must follow his own individual mistakes and successes in order really to profit thereby, and it is only upon a broad surgical basis that a study of these post-war results can be instructive; unfortunately such study of results was not possible to the individual surgeon during the active war period nor subsequently when the cases were beyond his reach. All the cases reported are taken from our surgical clinic at the U. S. Public Health Service Hospital No. 35, St. Louis, Mo., covering the period between July 1, 1919, and June 30, 1921.

NERVES

Hospital No. 35 was organized rather late to get nerve injuries requiring suture; nevertheless, from our observation of end-results we are convinced that the successful nerve suture must be end-to-end anastomosis and that each severed end before suture must show normal fasciculi.

We have found it necessary to resect neuromata from the ulnar nerve in four cases, twice each in the upper arm and forearm; in each instance an incision was made through the centre of the neuroma, the ends sliced off until normal fasciculi were found, when an end-to-end suture was done, being careful not to twist either trunk. In one case it was necessary to acutely flex the wrist in order to bring the ends together, and in another this was made possible only by transplanting the nerve to the front of the condyle of the humerus. In each case we surrounded the suture line with fat or muscle; whether or not this is worth the time and trouble, we are not prepared to say.

It is rather too early to give end-results, but in no case when last seen

had the thumb adductors filled out to normal. Three of these injuries followed gunshot and one incised wound.

We have removed painful neuromata from amputation stumps fourteen times; in five from finger stumps; once each from the median, musculospiral, and radial; twice each from the anterior tibial, posterior tibial and ulnar nerves.

At first we made a practice of injecting the trunk of the nerve with alcohol to prevent further neuromata, but in many cases the resultant pain was so severe and persistent that we abandoned the method and now simply cut the nerve off as high as possible above its end.

In three cases we had to free nerves from scar tissue: An ulnar in the forearm just below the humeral condyle, causing severe pain and numbness and twice previously operated on, was freed for three or four inches and then surrounded by a free fat transplant, with immediate relief from pain; a perineal was freed from scar tissue near the fibular head, with relief from pain; a sciatic was operated two and one-half years after injury, with excellent results. In this case the patient had been injured in the Argonne, November, 1918, by shrapnel in right gluteal region; the femur had been fractured; control of leg and foot had been lost and later trophic ulcers and eczema had occurred on the external side of the leg; the knee had become partially ankylosed. At operation we found the sciatic nerve bound to the greater trochanter by very dense scar tissue. The nerve was freed, scar tissue removed from the trunk and then surrounded by a free fat transplant. In eight days the ulcers of the leg had healed and sensation returned to the external leg and toes; the patient is still improving.

KNEES

The knee has been the most frequent of the large joints requiring surgical attention. It has been necessary in this series to open the knee-joint twelve times, with no subsequent infections.

For the removal of "joint mice" and semilunar cartilages the Jones semilunar incision has proven the most practicable; the longitudinal splitting of the patella gives an excellent exposure of the joint in front of the crucial ligaments, but if there be a foreign body behind these it is necessary, as in one of our own cases, also to split the capsule transversely; the large curved transverse incision likewise gives excellent exposure, but occasionally leaves a relaxed patellar tendon. We have found it best to control hemorrhage as the operation progresses and to change gloves just before entering the joint and immediately after the towels are clipped to the skin edges; all sponges being handled by forceps.

All the cases gave a history of injury. It may be generally stated that the longer the period between the injury and the operation, the greater the pathological changes present and the less chance of complete recovery.

One knee which had previously been operated and had been stiff and

draining over a year and also thought to be tuberculous, immediately healed and completely recovered all motion, by simply removing a large sequestrum from the patella.

We have had to amputate one thigh because of a destructive process in an old resected joint. So far we have resected only one knee-joint as a result of injury and it proved to be tuberculous.

We have removed seven loose semilunar cartilages and eight " joint mice." The chief symptoms were pain, weakness, locking and sudden giving way of the joint. The X-ray was a great aid in diagnosis. All cases gave a definite history of injury.

The most instructive and satisfactory operation upon the knee was as follows: E. B. fractured his right patella October 15, 1919, and it was wired November 17, 1919. He was in bed three weeks. On February 2, 1920, in attempting to board a street-car he ruptured the wire sutures and separated the fragments; he was operated the second time February 11, 1920, when the joint became infected, and he was in bed ninety days; the knee drained six weeks and complete ankylosis followed. The patient visited several large clinics seeking relief; an attempt was made at one hospital to bend the knee under general anæsthesia. After admission to our wards, X-ray showed complete bony ankylosis. On October 15, 1920, we performed the following operation: Longitudinal external and internal incisions joined just below the tibial tubercle; no joint could be found; the bone was chiseled about three-fourths through and then broken; condyles were then shaped, the internal the longer, and excavations made in the tibia to receive them, leaving a large spine between; a large pedunculated musculo-fascial flap was then sutured in place to cover the new joint surfaces; the quadriceps tendon was then severed by a Z-shaped incision and lengthened so that the two parts of the patella could be wired together; another pedunculated flap was then sutured under the patella and the wound closed. A posterior splint with extension was applied for seven or eight days when very gentle passive motion was begun. Personal attention was given to this case for six or eight months. The patient at present has a useful and strong knee-joint; he can raise his weight up and down on this limb alone, walk up and down stairs, dance, etc.; he has 48-degree motion, with a good chance of greater range of motion in the future.

Other Joints.—We have successfully made a metacarpo-phalangeal joint for the index finger in one case and the ring finger in another; the scaphoid has been removed to obtain more motion in a wrist and one elbow-joint has been resected. Amputations following old joint injuries will be mentioned later.

Recurrent Inguinal Hernia.—Recurrent hernias may be considered as sequel injuries. We have no fair basis upon which to figure the percentage of these recurrences. In the two years covered by this report we have operated three hundred twenty inguinal hernias and of these fourteen were

recurrent following operation elsewhere and therefore do not properly belong to this same series.

As is the rule, the direct hernias had recurred most frequently. Three were double with recurrence on both sides and one was a double with unilateral recurrence.

In one recurrent on the right side we found the stump of an appendix which had never been invaginated and still produced symptoms. One case had a fecal fistula caused apparently by a needle puncture of the bowel, and it required several attempts before we were successful in closing it. In three cases very large sacs had never been removed and in one case the incision had been made only through the skin. One undescended testicle was found bound in a mass of scar tissue; in one case both testicles were undescended and it was necessary to replace one of these in the abdomen to relieve pain.

Other Hernias.—It was necessary to repair the anterior abdominal wall twenty-four times and the lumbar once. In practically all the abdominal hernias, adhesions of the bowel or omentum were found and freed. In twelve cases the hernia followed drainage of an appendix; in one, as a result of gall-bladder drainage, we accidentally opened up the old tract and soiled the wound with bile, but this did not prevent primary union and an excellent result.

We have used the Mayo overlapping method and have not hesitated to cut large flaps from the anterior recti sheaths when needed, always suturing the remaining edge of the sheath to the muscle to prevent a muscle hernia. We have used chromic catgut No. II in all cases.

The patient with the lumbar hernia was wounded October 27, 1918, by a high-explosive shell and was in the hospital until discharged from the Army. The back muscles on the right side between the ribs and ilium were shot away and only thin skin covered the bowel, allowing the cæcum to be picked up in the hand. In July, 1919, we freed the cæcum from the skin, replaced it in the abdomen and transplanted fascia lata from the thigh to the back; this with the scar tissue present gave him a strong and useful back free from pain.

Muscle Hernias.—Débridement no doubt saved many lives and many limbs; this, however, was the chief cause of muscle hernias, which, in our experience at least, is a new cause of post-bellum disability; we believe that muscle hernias were rather rare before the war.

Weakness and pain are the chief subjective symptoms; objectively the diagnosis is easy and treatment is simplicity itself. The edges of the fascia should be well freed before suture.

In clean cases when it becomes necessary to remove for transplanting a part of the fascia over a muscle, hernia can be prevented by suturing the remaining edge of the fascia directly to the muscle.

We have repaired twenty-three muscle hernias in sixteen patients. It is interesting to note that eleven of these patients received their primary wounds

in the Argonne. All the hernias were of the muscles of the lower extremity. The fascia lata over the external thigh was repaired eight times; over the quadriceps, nine times; over the external belly of the biceps femoris once; over the tibialis anticus three times and the lumbar fascia twice. The back cases were most extensive; here the fascia had been split transversely across both sides and part of it removed; in each were foreign bodies requiring removal and in one case the erector spinæ had been severed and partly removed.

Foreign Bodies.—Many symptomless foreign bodies were located which we did not molest, while a few were removed for the psychic effect. One in a silent area in the brain, one in or near the heart with no symptoms, one in the centre of the liver, and two in the lungs we thought best to respect and leave alone on account of the seriousness of the operation involved.

In only two cases were we unsuccessful in removing the foreign body. All were previously located with the X-ray and upon several occasions it was necessary to use the fluoroscope during the operation. Local anæsthesia can be used in practically all cases of foreign body. This gives the operator the advantage of leisurely searching for hours if need be and with the coöperation of the patient.

The majority of the foreign bodies have been high-explosive shell fragments or bullets. We have found gauze packing, wood knife-handle, etc. Practically all foreign bodies were claimed by the patient as souvenirs, which prevented our making a collection. We have removed one or more foreign bodies from thirty-five patients as follows:

High-explosive shell fragment	24
Bullets	7
Wood (knife handle)	1
Gauze drain	1
Piece of emery wheel	1
Needle	1
They were removed from the	
Head and neck	6 times
Upper chest	2 times
Upper extremity	5 times
Body	8 times
(Of these seven were removed from the posterior region and two from in front of the sacrum.)	
Lower extremity	6 times
(One each from tibia and fibula.)	
Near shoulder joint	4 times
(Two under clavicle near ribs; one from spine of scapula and one in front of body of scapula.)	
Place not mentioned	4 times

Fourteen of these patients were wounded in the Argonne and fifteen had been operated previously.

Skull Injuries.—Considering the number of skull injuries seen, only a few required operation.

We are convinced that bone grafting to cover defects of the skull is not a proper surgical procedure. A bone graft requires stress in order to be a success. A bone graft of the skull lives about two years, either becoming absorbed or its edges becoming rarified, and requiring removal on account of irritation. Cartilage seems to be the proper material to use; it is easily obtained, shaped and sutured to the periosteum over the defect and upon the edges of the bone instead of the dura. Coughlin, of St. Louis, has proved such cartilage transplants live in the skin for twenty years. The chief symptoms relieved are dizziness and throbbing headache.

We have grafted cartilage over skull defects in three patients; two of these had been bone-grafted nearly two years before; the other had a large defect, with scalp adherent to dura. Two of the patients were epileptics, but the convulsions were not much relieved, although the headaches and dizzy spells were alleviated.

One skull injury presented a pulsating tumor over the longitudinal sinus following a high-explosive shell wound; at operation this proved to be a cirsoid aneurism of the temporal artery, which we removed.

An attempt to drain an abscess of one year's duration was followed by meningitis and death. The interesting part is that the abscess was not suspected and was discovered at operation.

Other Bone and Cartilaginous Grafts.—We have used only autogenous grafts and the tibia has been the favorite source. We prefer the inlay method. Kangaroo tendon and chromic gut are preferable to beef-bone screws, although the latter are more easily handled when properly made.

The cases requiring bone grafts have been difficult and had previously been operated with much resultant scar tissue, etc. We have had to graft two humeri, two ulnæ, one radius, one superior maxilla and three spines, following injury.

One patient, a great friend of John Barleycorn, refractured a united medullary graft of the humerus and obtained another false joint. In another case it was necessary to remove one bone graft of the humerus which had failed to unite; good approximation of the ends was obtained, but the patient left the hospital against advice and has failed to report back as promised. (Another friend of John Barleycorn.)

One of the ulnar grafts bridging a two and one-half-inch defect became infected; nevertheless the graft lived and new bone united the ends, using the graft as a bridge and growing along its side. A false joint following a fracture of the ulna and radius very near the elbow-joint was especially difficult but successful.

The three bone grafts of the spine were successful and the patients are up and about again. We did not split the spinous processes, as in the Albee operation, but denuded the left sides completely and fastened the graft to the denuded side of the spines with kangaroo tendon through drill holes. Body casts, split down the sides and known before operation to be comfortable, were applied at once and worn for months.

The cartilaginous graft of the superior maxilla was especially instructive and gave an excellent result. A piece of cartilage was removed from the chest, shaped like a tennis racket, the handle for the zygoma, and then sutured to the remaining root of the zygoma and edge of the orbit through drill holes; the scar tissue and skin were then sutured over it. Later the ear was built up by a cartilaginous transplant and the boy departed, happy that he could again associate with his friends without embarrassment.

Correction of Deformities.—Deformities resulting from injury have been corrected for eleven patients. One of the worst deformities of the leg we have seen followed fracture of tibia and fibula at the junction of middle and lower third, which had been operated, a Lane's plate applied and the patient had been permitted to be up too soon. The leg was Z-shaped and the patient in an undesirable psychic state because he had been "turned down" at several hospitals and at the "largest clinic in the United States." An open osteotomy, with nearly a year's personal attention, gave him a leg, as the patient writes, "surpassing our most sanguine expectations."

We had two chronic dislocations of the shoulder-joint: one, a blacksmith, has been following his trade over a year without recurrence, who was treated by simply imbricating the capsule; in the other, which would dislocate even when patient sneezed, it was necessary to split the capsule and overlap it. The anterior incision was used with chromic gut No. II as suture material in both cases.

Tenotomies of extensor toe tendons were done four times and of the tendo Achillis three times. A deformed metacarpal, following fracture, was corrected by open osteotomy and extension; and a cervical rib removed in a patient previously diagnosed at different hospitals as having neurasthenia, psychoneurosis, paraplegia, rheumatism, pellagra, chronic malaria, bone tumors of the back, syphilis and osteitis; the X-ray soon revealed the actual trouble.

Stumps.—During this period we have had twenty-five stumps to repair or reamputate on account of pain and non-healing and in order that artificial limbs might be comfortably worn. This occurred in the leg eleven times, thigh five times, forearm four times, arm two times, fingers five times. Fourteen of these patients complained of pain; three had an osteitis of the ends of bones; seven had spurs on the ends of bones; one had a foreign body and one a piece of silkworm gut; seven had painful neuromata; six had ulcerated stump; three had too-short skin flaps; three had painful scars, requiring removal.

Post-operative Adhesions.—Had we operated all cases diagnosed as peritoneal adhesions our list would, figuratively speaking, be legion. Nevertheless, in a few cases the diagnosis has been definite and operation has relieved the symptoms. Eight of the cases followed appendectomy; in all, the chief symptom was pain; one had not been free from pain for two years. The cæcum and omentum were involved in all; there was a hernia in three; a stump of an appendix was found in one.

One case had an obstruction of the bowel following an abdominal bullet wound. We found a band the size of a lead pencil reaching from the anterior abdominal wall to the spine with a loop of intestine about it; a band of omentum was also found fastened to the sacrum and two loops of bowel "hooked" together by adhesions; it was only by the freeing of this last that we were able to release the gas and it was not necessary to resect.

Another abdomen was injured by a horse falling upon the patient; the spleen was ruptured and appendix was removed. The patient suffered greatly and later another operation was done for adhesions and hernia, but this had not relieved him. We found the cæcum and ileum bound to the anterior abdominal wall and the ileum bound to the cæcum in such a manner as to cause an acute kink at its junction with the large bowel. The adhesions were freed and the hernia repaired, with complete relief.

Another abdominal injury had been diagnosed psychoneurosis on account of the neurotic symptoms. We found the omentum and transverse colon bound to the liver. They were freed and replaced in proper position, with relief to the patient, who is now again following his profession as a dentist.

Scars.—One would expect many disabling scars in this class of patients, but this has not proven true in our clinic.

We have freed the flexor tendons of a wrist, the biceps tendon of an arm, also scar tissue about an elbow preventing motion and once the shoulder-joint, preventing abduction of the arm.

Painful scars have been removed from the first two fingers; from over the greater trochanter where a free fat transplant was made; from the groin in which a tuberculous stump of a spermatic cord was found and removed; from over the tibia in two cases and from the dorsum of the foot in one. In a painful scar of a herniotomy wound we found a silk suture causing the trouble. In one case a nerve was caught in a wound of the thigh. One case was unique: The patient suffered an X-ray burn of the heel and tendo Achillis when his ankle was examined for fracture; this had left a painful ulcerating scar which would not heal and had been unsuccessfully skin-grafted. After dissecting out the ulcer, we made a pedicle skin-graft from the opposite thigh to the heel, holding the parts in place for a week with a cast. The patient has been using the foot now for nearly a year: A short time ago we had the pleasure of seeing this graft, which still retained its pad of fat and hair was growing upon it. Skin grafts to be successful should be taken from above the knee or the elbow.

Amputations.—It has required a nicety of judgment to decide when and when not to amputate, as the patients are frequently much more radical than the doctor. Many who have waited patiently in the hospital for months and who see their friends recover and leave are anxious to settle the matter at once, and some few have attempted to secure amputation elsewhere after being advised to wait longer.

In children who stand chronic infection better it no doubt pays to be ultraconservative, but the adult who has fought infection for two or three

years and cannot be promised more than a slim chance of a fair limb in some two or three more years of hospital life with the added danger of organic changes in the liver, kidney, heart, etc., and who can be put back to a gainful occupation at once with an artificial limb (especially where his disability will be about the same under the most favorable outcome), is allowed to decide the question of amputation for himself. Amputation was chosen in all but one case.

We have amputated eight fingers and one toe on account of contractures and ankyloses; the thigh three times, once because of a rupture of the femoral artery during a dressing, once because of a destructive process in a resected knee-joint and once because of a chronic infection of the knee-joint; the leg, three times due to destroyed ankle-joints following infection; the arm and forearm once each; a shoulder girdle because of a sarcoma of the scapula following injury and a hip-joint with the head and neck of the femur destroyed. This last case was especially difficult and interesting as follows:

> B. K. received multiple gunshot wounds of the upper thigh and abdomen July, 1918, in the Vosges Mountains. A few foreign bodies were removed at once and in a few days he was sent to a base hospital and then to Bordeaux in August, 1918. On the train he had a severe hemorrhage and was transfused; seven days later was operated and the head and neck of femur removed and cast applied. He was sent home December, 1918, and operated again January, 1919, for a chronic osteomyelitis of femur and ilium. Was in the hospital five months, but the wounds did not heal. He was operated again in May, June and August, 1919, and then transferred to another hospital and operated again in September, 1919, but without improvement. He was then discharged from the army and sent to Chicago in October, 1919, and later to Hospital No. 35 in December, 1920, while still draining.
>
> X-ray examination showed the head and neck of femur gone and osteomyelitis of the ilium and greater trochanter with a foreign body in the pelvis surrounded by bismuth paste; the limb was shortened, motion limited, leg cold and indurated, muscles atrophic, skin shiny, knee and ankle ankylosed, and part of the brim of the pelvis gone. We amputated at the hip-joint February, 1921; on April 7, 1921, we removed the foreign body from in front of the sacrum and, on April 25, 1921, we cleaned out the acetabulum, both operations under local anæsthesia. The patient was immediately permitted to be out of bed, gained weight and was discharged. A small sinus still occasionally drains bismuth.

Osteomyelitis.—There has been no post-bellum condition following injury giving more concern and requiring more care and attention than osteomyelitis; and as the patients usually arrive at the hospital discouraged and often debilitated from long-continued infection, we know no class more pleased and appreciative when they go home cured unless it be the surgical staff.

The cure of osteomyelitis depends more upon unfaltering care and

attention after the proper "cleaning up" than upon any new and wonderful operation or method of treatment.

In general we have followed the simple plan of removing *all* diseased bone with mallet and chisel (no matter where the diseased bone, it should be followed to the bitter end and the cavity well saucerized), swabbing the cavity with iodine and packing with gauze. The wound is then daily dressed and repacked and permitted to heal from the bottom only. It has occasionally been necessary to skin-graft healthy granulations and, under favorable circumstances, we have been able to save much time by filling the bone cavity with muscle or skin.

We have been especially pleased in certain patients, where the general resistance was low, the wound sluggish and not responsive to treatment, to see them change as if by magic and the wound heal, by simply receiving a few doses of typhoid bacilli intravenously, beginning with 50,000,000 and increased according to reaction. We have used typhoid bacilli as a foreign proteid since 1913 and so far have had no occasion for regret, although upon two or three occasions we have been somewhat worried, and it is well, when starting a new course of injections after an interval, to begin very carefully.

We have operated one or more times upon fifty-two patients with chronic osteomyelitis following injury; all had been previously operated, one as many as fourteen times. In these cases the injury was caused by high-explosive shell fourteen times, by machine-gun bullet four times, by gunshot wound ten times, by horse kick two times, by bayonet wound one time, by external violence eight times, not mentioned thirteen times. In seventeen cases the bone was fractured. The tibia was involved twelve times, the scapula was involved three times, the metatarsal was involved three times, the fibula was involved one time, the femur was involved fifteen times, the humerus was involved five times, the skull was involved one time, the ilium was involved two times, the ribs were involved three times, the os calcis was involved three times, the ulna was involved one time, the radius was involved one time, the metacarpal was involved two times, the carpal was involved four times, the sacrum was involved one time. Twenty of these patients were injured in the Argonne. There was a hole through the bone eight times, Lane's plates were removed two times, other foreign bodies were removed three times, tuberculosis was suspected two times.

Miscellaneous.—It has been necessary to do one cholecystenterostomy following drainage of a gall-bladder of several months' duration; to correct a retroversion of uterus in one patient and to remove a cystic ovary with a repair of the perineum in another; the former was injured by a horse, the latter injury we have not been able to connect with service: We have removed a large fibroma(?) of the forearm following injury and explored a large post-peritoneal sarcoma following the removal of an injured testicle and have removed another injured testicle which proved to be sarcomatous; a large inflammatory tumor following gunshot wound of left knee was removed.

We have twice unsuccessfully attempted to repair the sphincter ani of one patient, which had previously been severed in three different locations.

One patient who gave the history of having fallen into a trench and injured his back and who was sent to the hospital, diagnosed inguinal hernia on account of a swelling in the right groin, was relieved of all symptoms by anchoring a floating kidney.

In the above superficial presentation of these post-war injuries it has been impossible to dilate upon the individual cases, some of which gave most interesting histories of what happened to them both upon the battlefield and later in the hospitals in France or as prisoners in Germany. It no doubt will be still more interesting in the distant future to be able to combine the army and navy records with our own and follow the cases to a ripe and happy old age.

THE TREATMENT OF SURGICAL TUBERCULOSIS WITH THE CARBON-ARC LAMP

By Paul Kurt Sauer, M.D.
of New York, N. Y.

In the early part of 1917, when the writer was in Copenhagen, his attention was called to the treatment of surgical tuberculosis at the Finsen Lysinstitut by means of the carbon-arc lamp. Dr. Axel Reyn, the director of the Finsen Institute, and Dr. N. P. Ernst had been treating this type of cases with great success for several years, and both were very enthusiastic in their support of this form of treatment. Some of the cases treated were of long standing and the results achieved truly remarkable. It is the purpose of this paper to give a short account of this form of treatment. Some of the cases were treated at the Lenox Hill Hospital where two lamps have been installed.

The lamp (see illustration) is a twin-arc lamp with a reflector, and the arcs connected in series. A current of twenty-five amperes and one hundred and ten volts passes through these carbons, thus giving each carbon half of the voltage. The carbons are not enclosed in glass but emit their rays directly on the patient. A special chemical-core carbon is used to produce the desired rays.

The treatment consists in placing the patient before the light and exposing the affected part and as much of the surrounding area of the body as possible. Since the effectiveness of the rays decreases as the square of the distance from the light, it is obvious that the nearer the patient is to the light the greater the benefit. As the skin becomes more deeply pigmented and the heat from the lamp is better borne, the patient can be quite closely approximated to the lamp. The time of the exposure varies with the age, sex, and pigmentation of the patient. As a rule an adult male of dark complexion can be started with a half-hour exposure. Children should not be exposed more than fifteen minutes at first. After three or four treatments, each a little longer than the preceding, exposures up to an hour and a half may be given. At least three treatments a week should be administered.

About eight or ten hours after the first treatment a deep erythema is noticed. This may be uncomfortable for a few hours, but not more so than a sunburn acquired while at the beach. If too long an exposure is given the first time blistering may result. After two or three treatments desquamation will take place and later pigmentation will result. While the patient is under the light his respirations become slower and deeper, but the pulse-rate and volume are not materially changed. There is no marked alteration in the blood count.

Marconi states that he noticed local sweating around the wounds in treating these cases, but I have not noticed this, as the patients perspire so pro-

fusely that the entire exposed surface is about equally covered with perspiration. The effect on wounds with sinuses is identical with that noticed by Lovett in the Thézac-Porsmeur method of treatment.

The carbon-arc lamp has several advantages over the natural sunlight in the treatment of these cases. The patients need not go to sanatoria at the seashore or to high altitudes in order to get the effective rays. (These rays are usually filtered out by the dust and smoke at low altitudes.) The weather need not be depended upon; for in this way no treatment days are lost. What is probably more important, however, the patient need not stop his work in order to get the benefit of treatment, and can still live at home.

The mercury vapor-quartz lamp, while more economical in operation, is more costly and not as effective. Reyn has tried this lamp without much success and has given it up in favor of the arc lamp. The rays from the quartz lamp are almost entirely of the ultra-violet end of the spectrum, not very penetrating, but highly irritating. The quality of the rays, furthermore, is not constant, as the lamp becomes weaker with usage.

The Röntgen rays, while perhaps just as effective in the treatment of tuberculous cervical adenitis where the glands have not broken down, is not as effective in the treatment of tuberculous sinuses. It is also much more dangerous and must be handled by a specialist. Even then, as Baisch reports, burns sometimes do not appear until years after the last treatment. There is no danger of burning with the arc light except insofar as the sun is liable to cause burns, and the lamp may be used by any one with absolute safety to himself and the patient.

REPORT OF CASES

A. H. W., waiter, fifty-four, was admitted to the Lenox Hill Hospital on March 23, 1917, complaining of a small lump at the right costal border. The tumor was firm, but slightly fluctuating, and not tender. At times slight pain is noticed in this area, and the patient is not able to sleep on that side. The tumor has been getting larger for the last ten months. Except for pneumonia at the age of twelve and again at fifty, and typhoid at the age of twenty-two the past history is negative. Venereal infection denied. Except for the tumor the physical examination was negative. On March 24, 1917, about two inches of the cartilaginous and osseous portions of the eighth rib were resected. (Pathological diagnosis: Tuberculous osteitis of the rib.) After three or four months the wound closed. In the latter part of September the wound reopened, and was not healed until June, 1918. In March, 1919, the wound again opened and he was admitted to the hospital on December 12, 1919, as the wound had not healed up to that time. Reoperated on December 17, and on February 6, 1920. At this operation portions of the ninth, tenth, and eleventh ribs were resected. On June 11, 1920, the light treatments were begun. At that time two sinuses each about four inches in length were present. One opening was just to the right of the xiphoid cartilage and the other about an inch toward the nipple line. Rough bone of the sternum was easily felt with a probe. Blood count showed red blood-cells 4,330,000, white blood-cells 7400, hæmoglobin 90 per cent., polymorphonuclears seventy-four per cent., lymphocytes twenty-six per cent. The urine was negative. On July 21 the median fistula has healed. In September the patient claimed his general well-being was better than it had been in several years, that he had no more pain at

the site of the fistulas, and that he could now sleep with comfort on that side. The last fistula was healed on October 12, 1920, and he was discharged from treatment on December 15, 1920. When last seen in July, 1921, he had had no recurrence of any symptoms.

J. F., orderly, forty-nine, was admitted to the Lenox Hill Hospital on June 10, 1918, with an abscess of the left side of the neck. Operated upon June 13 for tuberculous cervical adenitis with abscess formation. A small draining fistula persisted when he was discharged in July. He was again operated upon in November of that year as the fistula had not healed and another abscess had formed. At this operation most of the glands were removed, and he was discharged with a draining sinus in February, 1920. When he was first treated with the arc light in October the sinus was not yet healed and a slight amount of secretion persisted. Just behind this sinus at the anterior border of the sternomastoid muscle a small group of glands about the size of a walnut was noticed. There was some pain in this region associated with movements of the head, and some tenderness on pressure. In December, 1920, the patient stopped treatments but resumed them again in February, 1921. In March the mass had disappeared, the pain had ceased, and the sinus was completely healed. When last seen in July, 1921, the patient had had no recurrence of symptoms.

R. H. W., secretary, forty-seven, was operated upon on November 30, 1920, at the Lenox Hill Hospital for a left tuberculous kidney (nephrectomy). After a stormy convalescence she was discharged from the hospital on January 30, 1921, with three draining sinuses. The sinuses were very slow in healing and light treatments were begun on March 12, 1921. On May 9 the patient discontinued treatments. At that time two fistulæ were completely healed and a small scab had formed over the third fistula. The drainage from the last fistula was negligible. The patient wrote in July that the fistula had not yet closed.

W. G., clerk, thirty-nine. For the past five years the patient has been suffering from tuberculous sinuses on the anterior chest wall, one or more of which have been open all the time. About three years ago the axillary glands became involved to such an extent that they broke down and five sinuses made their appearance. On July 13, 1918, the sinuses were curetted and packed with iodoform gauze. The pathological report confirmed the diagnosis of tuberculosis. The X-ray showed no bone involvement. On physical examination slight dulness and a high-pitched percussion sound were noted over the left apex, anteriorly and posteriorly. The left pectoralis muscle is more prominent than the right, is stiff, and has a wooden feeling. There are three sinuses in the mid-clavicular line, one at the level of the second rib, and two at the level of the third rib. In the left axilla there are five fistulæ. The urine is negative. Red blood-cells 4,350,000, white blood-cells 9600, polymorphonuclears sixty-two per cent., small lymphocytes thirty-one per cent., large lymphocytes six per cent., basophiles one per cent. This patient began light treatments in June, 1920, and has not responded to the treatment except that one fistula in the axilla is healed. He claims that he feels somewhat better generally, but as his mentality is of quite low grade his statements are unreliable.

J. B., watchman, forty-two. Admitted to the New York Skin and Cancer Hospital on February 8, 1921, complaining of a small, slightly painful and tender lump on the right side of his neck along the anterior border of the sternomastoid muscle, of seven months' duration, which was gradually increasing in size. Has had no night-sweats, but has been hoarse for the last four years, and coughs moderately in the mornings. About six years ago the glands on both sides were removed. After an operation for a tuberculous abscess he was discharged with an open sinus on February 19, 1920. He was readmitted on April 25, 1921, with an oval-shaped ulcer and a discharging sinus at the site of the previous operation.

FIG. 1.—Twin-arc lamp.

Operated upon again and the sinuses curetted and packed. He was discharged with an open wound on May 2, 1921, and began the light treatments at the Lenox Hill Hospital on May 5, 1921. By the end of June the wound was entirely closed and the patient was free from all symptoms. Notwithstanding repeated admonitions to continue the treatments the patient has stopped coming.

M. H., office worker, twenty-one. (Referred by Dr. Nathan Green.) Admitted to St. Luke's Hospital on June 4, 1920, complaining of swelling of the abdomen. Operated upon by Doctor Green for tuberculous peritonitis, and fluid evacuated. Red blood-cells 3,600,000, white blood-cells 7900, polymorphonuclears sixty-two per cent., lymphocytes thirty-eight per cent. Urine acid, 1030, faint trace of albumin, a few casts, and some white blood-corpuscles. She was discharged from the hospital on June 19th, with four sinuses in the operation scar. From then until March, 1921, she was treated in the out-patient department, and with X-ray without any material benefit. In March she began light treatments, and noticed that in June she was free from all pain. One sinus (the lowest one) had healed. July 12 all sinuses were healed and the patient was discharged on August 9, 1921. She has had no more pain since June and has gained sixteen pounds since beginning the light treatment. There has been no accumulation of fluid in the abdomen.

A. McC., housework, seventeen, came to the out-patient department of the Lenox Hill Hospital in August, 1920, complaining of ulcers on her anterior chest wall, of two years' duration. Various forms of treatment had done no good. One ulcer was over the sterno-clavicular joint and two others below along the left border of the sternum. X-ray showed no bone involvement. Wassermann was negative. The axillary glands were not palpable. Urine normal. Blood count showed nothing of any moment. After one hundred and forty exposures all three ulcers had healed and the patient was discharged.

TABULATION OF CASES

Case No.	Initials	Sex	Age	Diagnosis	No. of treatments	Total hours	Results
1	A. H. W.	M.	54	Tbc. osteitis	70	54	Cured
2	J. F.	M.	49	Tbc. cervical adenitis	53	39	Cured
3	R. H. W.	F.	47	Tbc. kidney	25	16	Improved *
4	W. G.	M.	39	Tbc. axillary adenitis	160	108	Unimproved
5	J. B.	M.	42	Tbc. cervical adenitis	24	18	Improved *
6	M. H.	F.	21	Tbc. peritonitis	40	30	Cured
7	A. McC.	F.	17	Tbc. of chest wall	140	105	Cured

* Patient stopped coming for further treatments.

CONCLUSIONS

1. The carbon-arc lamp is an effective agent in curing cases of surgical tuberculosis.

2. It is as effective as the natural sunlight and has the advantages of convenience and independence of the weather.

3. It is just as effective, if not more so, than the X-rays without the attendant dangers.

4. It is far more effective than the quartz-mercury vapor lamp, as has been amply demonstrated by Reyn.

TUBERCULOUS ABSCESSES OF THE CHEST WALL*

By Hugh Auchincloss, M.D.
of New York, N. Y.

Nine cases are presented in this review. One case, a Chinaman, died shortly after admission to the hospital. One case, but recently admitted, has not been operated on, and is presented as a clinical example of the subject and not a result.

Some of the case histories are very long. In reporting them, an attempt has been made to focus on the chest wall condition, and cite as briefly as possible the other associated lesions, except where these lesions are of unusual interest. The ages of the patients varied from four and a half years to sixty-two. The duration of abscess was from two weeks to eight months.

Symptoms: A soft swelling on the chest wall, possibly preceded by a period of slight discomfort, slightly tender to pressure, fixed to the deeper parts, sometimes beneath the pectoralis major, its superficial portion somewhat movable when the muscle is relaxed, but firm, and fixed when the muscle is taut. It is usually evidently fluctuant.

The general health is below par; there may have been loss of weight, the picture may be that of an associated condition. There is some secondary anæmia with no leucocytosis. There may be little or no temperature rise. The X-ray may show dense shadows beneath the lesion, and sometimes evidence of calcification.

Location—usually in the anterior axillary line, low down—in the third interspace close to the sternum, or beneath the pectoralis major muscle. Seven of the nine were on the left side. The two on the right side were associated with Addison's disease, probably a coincidence, though their X-ray plates are strikingly similar.

Etiological Factors: Three cases were associated with cervical and axillary lymph-node enlargements. Seven cases gave a history of either influenza, or some form of respiratory disease, the description of which resembled pneumonia or a pleurisy with effusion, or encapsulated empyema. Four, and possibly five, had pulmonary tuberculosis.

Pathology: An attempt was made to prove the rib to be the distributing focus for the abscess. It was thought that this would prove to be the case in most instances. It is impossible to state that it may not have been in these cases and not discovered. Ribs were found tuberculous in two of the cases, but only after they were removed at secondary operations. Staining inequality in the costal cartilage of one case was found. In no case was rib removed at a primary operation found to be tuberculous. No contention is made that such is the rule, for it is well known that a rib may act as such a dis-

* Read before the New York Surgical Society, October 26, 1921.

tributing focus. On the other hand, the assumption that a tuberculous abscess of the chest wall is nearly always from a "tuberculous rib or sternum," an assumption that is rather general, is probably untrue.

Six abscesses were of the "collar-button" or "dum-bell" variety. A subcutaneous or submuscular abscess communicated by a narrow sinus through an intercostal space with a cavity or cleft beneath the intercostal muscles, and their adjacent ribs between them and the lung. This deep space corresponded to the pleural layers. Four cases that were cultured showed sterile fluid and positive guinea-pig inoculations. One pulsating empyema necessitatis grew out hæmolytic streptococci and contained tubercle bacilli that were recovered from a guinea-pig. One case was not cultured but had a positive guinea-pig inoculation. One case showed tuberculous granulation tissue but no cultures nor guinea-pig injection were done. Very extensive calcification of the pleura occurred in the two cases of Addison's disease. In one of these tubercle bacilli were recovered from a guinea-pig, whereas a diagnosis of chronic inflammation and not tuberculosis was made from the tissues of the abscess wall. In the other case the wall had probably been the site of an old localized empyema that had healed with extensive calcification and absorption of the abscess. This was the Chinaman with extensive tuberculosis of both adrenals, and whether the lesion in the chest wall was tuberculous or not remains somewhat in doubt. This Case III and Case IX † are the only ones in doubt, however, and Case VII is included because of its striking similarity to Case VIII.

Associated Conditions of the Nine: 1. Enlarged cervical and axillary nodes. 2. Enlarged cervical nodes removed one year previously. 3. Chronic pulmonary tuberculosis. Tuberculous empyema found after his second operation, but quite possibly latent previously. 4. Hæmolytic streptococcus pulsating empyema necessitatis following right upper and left lower lobe pneumonia; pulmonary tuberculosis. 5. Pulmonary tuberculosis; tuberculosis of elbow joint; tuberculous peritonitis; Pott's disease; tuberculous psoas abscess. 6. Tuberculosis of axillary lymph-nodes. Addison's disease; tuberculosis of mesenteric lymph-nodes. 8. Addison's disease. 9. Pulmonary tuberculosis; syphilis.

Rationale of Treatment: Surgery can be of real value in these conditions, but it will fail without certain precautions.

1. Before operating the general condition should be improved as far as possible.

2. Associated lesions should be looked for and if pulmonary tuberculosis is found, as much as can be done should be done under local anæsthesia.

3. As much of the abscess wall as possible should be excised by clean dissection and as much tubercle tissue removed as is practical. The principle to be followed is to bring as much uninfected and well-nourished tissue as possible in apposition on closing the wound. Such rib excision should be

† Since proven tuberculous.

done to expose an intercostal sinus and its underlying cleft or cavity and the walls of the latter excised gently but as completely as possible with a sharp curette. A small amount of iodoform powder may be smeared over the remaining wall.

4. The dead spaces should then be obliterated as far as possible. This is difficult in the deep cavity, but is aided by rib removal, and in the cases beneath the pectoralis major the origin of the pectoralis minor from the ribs may be severed and the muscle fibres, with their blood supply preserved, inserted into the cavity. The pectoralis major is sutured where it has been split and the skin incision closed either completely, or if there is much oozing, with small drains just through the skin. Blood is expressed from the wound as far as possible and pressure applied over the dressing by a large chest adhesive strapping and a temporary snug bandage outside. Every effort should be made to secure primary closure, and it is well, if possible, to avoid dressing the wound for twelve to fourteen days unless otherwise indicated.

A persistent course of anti-tuberculous treatment should then be begun and carried out for months and years.

CONCLUSIONS

1. Tuberculous abscesses of the chest wall are frequent enough to be of importance to the general surgeon, yet rare enough for many surgeons not to have had enough cases for study as to their pathogenesis and treatment.

2. There is a widespread opinion that such cases are, in the majority of instances, due to a " tuberculous rib " as the distributing or " primary " focus. The cases studied would indicate this not to be the case. The ribs did not show tuberculosis except after pieces of them had been removed and continuation of the tuberculous infection had occurred with secondary infection.

3. The distributing focus for the abscess seems to be from those structures immediately beneath the bony, cartilaginous or muscular chest wall. The lungs, the pleura and the mediastinal lymphatics seem preëminently responsible. The abscess is frequently deep as well as superficial to the chest wall; *e.g.*, the " collar-button " or " dum-bell " abscess.

4. The abscesses occur chiefly on the antero-lateral aspects of the chest wall rather than posteriorly.

5. The associated tuberculous lesions are varied in number and importance. They may or may not be more important than the abscess.

6. An extraordinarily large amount of calcium deposit may be present. The X-ray plates showing calcium are rather characteristic.

7. The story of influenza or an acute pulmonary condition may determine the onset of the abscess formation.

8. As complete excision of the tuberculous focus, leaving vascular, well-nourished walls to come together, with filling in dead spaces by muscle and pressure bandage and primary closure of wound is the treatment, though this may have to be modified by an associated lesion.

CASE RECORDS

CASE I.—E. M., four and one-half years; American. Admitted January 14, 1914, Ward 1. History: One year breast fed. At two and one-half years—treated O. P. D. P. H. three months for "trouble with leg" (no further record). Able to walk at three years, ten months—Manhattan E. and E., sore over left eye two weeks ago, lump on chest, painless, gradual increase in size. No cough, fever, loss of weight or appetite loss. Patient doesn't look sick. Left axillary glands slightly enlarged. Corneal opacity, small, right and left. Carious teeth. Left thigh 1 cm. smaller than right. Motion O. K. Tonsils O. K. Lungs clear. Spleen negative. Liver 2 cm. below costal margin. Wassermann negative. Von Pirquet positive.

Surgical condition: Third space left of sternum—3 cm. smooth hemispherical swelling, red, thin skin, fluctuating felt movable on deep pts.

Operation June 20, 1914: Doctor Jameson—two-inch incision, two cavities three-quarters inch in diameter. Sinus from each into mediastinum through intercostal space, thick yellow pus. (No excision.) Probed, swabbed with tincture of iodine. Silkworm gut closure tight. Pathological report—tuberculosis.

Discharged January 30th to country—wound unhealed on account of small stitch abscesses.

Follow-up October 25, 1921: Six and one-half years after operation. Wound closed three weeks after discharge and remained healed. Boy looks and acts well, but not very robust. There are enlarged nodes in both axillæ and both sides of the neck, those on the left side being about twice the size of those on the right.

Comment: A successful result. It seems scarcely possible that this case could have had a tuberculous bone focus, yet there was a cavity behind the intercostal muscles.

CASE II.—T. M., sixteen years, United States. June 24, 1915. History: Five months lump to left of sternum; first noticed it on throwing a ball. Slightly tender and reddened for a week—never persistently painful. Aspiration two weeks ago—pus obtained, recurred in three to four days. One year ago glands removed from the left side of neck. Six years ago double inguinal hernia operation. No cough; night-sweats; history of tuberculosis in family or associates. Physical examination—pyorrhœa and caries. Scar in neck. Slight enlargement cervical nodes—axillæ free. On anterior chest, third to fifth rib left side, soft fluctuating swelling which is not tender. Skin reddened and thin toward median line. Sputum and urine negative for tuberculosis.

Operation June 26, 1915: Abscess over sternum at level of second, third and fourth costal cartilages and beneath pectoralis major to its margin near axilla containing greenish-white pus with floccules in it. Wall—bluish granulation tissue. Beneath intercostals in second space was a small pocket that could not be seen communicating with any manifestly inflamed bone.

Elipse of thin skin removed; cavity entered after much of the abscess had been dissected away. Small amount iodoform sprinkled into deep pocket between ribs. Muscle sutured and skin sutured with few twisted silkworm gut strands at lower angle. Pressure dressing.

Pathological report: Tuberculosis. Smears—no tubercle bacilli found. Guinea-pig injection—pig developed tuberculosis.

Discharged on sixth day. Wound healed without inflammation; slight discharge from drainage tract.

Follow-up, July 25, 1915: Gained five pounds. Tiny drainage spot granulating, now flush with skin.

October 25, 1921: Six years, four months. Has enlisted in army; sixth year is up April, 1922. Is now a corporal of Mounted Police Guard, Fort Sam Houston,

Texas. Weight 144 pounds. On football team; is sharp shooter. Also won blue ribbon in horse race. After coming home never was ill. No cough.

Comment: Successful result. No evidence of bone focus. Cavity behind the intercostal muscles.

Case III.—E. F., fifty-six years, cigarmaker, Cuba; in United States twenty years.

Always a heavy smoker, a cigarmaker by trade, and generally in good health up to five years ago, when he was in the French Hospital for a week with fever, cough and pain in his chest. "One-half a gallon" of fluid was taken from his left chest, and he was told he had pleurisy.

He felt quite well after this, maintained his weight, had no cough, sweats, hæmoptysis nor other symptoms indicative of tuberculosis until one month before entering the Presbyterian Hospital in May, 1918. He then noticed a lump "the size of an egg" in the lower part of his chest in the anterior axillary line at the level of the seventh rib. It didn't seem to grow but was slightly tender and gave him dull, aching pain. At the left apex were a few subcrepitant râles and dulness with diminished breath sounds. At the left base were occasional râles, dulness and diminished breath sounds. The swelling over his seventh rib anteriorly was soft and fluctuating. The skin over it was of normal color. Pus mixed with blood was obtained on aspiration and tubercle bacilli subsequently obtained from the lesions in the inoculated guinea-pig. Sputum which was very scant showed no tubercle bacilli. A tuberculosis fixation test, it is interesting to note, was negative.

X-ray showed increase in density at the left base "suggesting fluid, and increase in density at the left apex and accentuation in the linear markings in the right upper lobe" suggesting tuberculosis.

On June 12, 1918, he was operated on for the abscess. A cavity lined with bluish red granulations and containing about 100 c.c. of necrotic tissue and grumous fluid was dissected out. No evidence of rib involvement could be made out, and it appeared that the abscess lay almost entirely superficial to the intercostal muscles. Abut three inches of the sixth and seventh ribs were removed, together with the intercostal muscles. The underlying tissues seemed healthful and the skin was closed without drainage. A compression dressing was used to obliterate the dead space.

Pathological report showed "tuberculous granulation tissue." Through a misunderstanding the wound was dressed on the seventh day. The intention was to maintain a snug pressure for twelve to fourteen days. The wound was reported clean and apparently healed. He was discharged to the Out-patient Department. Within a few days a little fluid reaccumulated. A small hole was made and some thick, bloody fluid evacuated on the thirteenth day, and pressure reapplied with each dressing.

On the thirty-first day the wound was healed and stayed closed until five and one-half months after the operation. Two tiny sinuses, discharging almost nothing, then appeared, and scabbed across and reopened for ten months. I then referred the case to Doctor Hanford, who was then concentrating his attention on the treatment of tuberculous lymph-glands and chronic tuberculous sinuses. Following a course of X-ray treatment, the sinuses closed for a few months but reopened nineteen months after operation. Twenty-one months after operation he was readmitted to the hospital under Doctor Hanford's care.

March 16, 1920, the sinuses were explored, curetted and packed with five per cent. iodoform glycerin emulsion and further radiotherapy was instituted, and he was again discharged from the hospital. Twenty-three days later, following a considerable increase in the amount of discharge for a few days, it was found that a probe be passed into a larger cavity, presumably into his pleural cavity.

SUMMARY

	1. E. M.	2. T. M.	3. E. F.	4. A. N.	5. S. W.	6. G. P. K.	7. W. L. C.	8. F. M.	9. D. M.
Age	4 1/2	16	56	19	30	33	53	37	62
Nationality ...	U. S.	U. S.	Cuba	U. S.	W. Indies	U. S.	China	Italy	U. S.
Duration of abscess	2 wks.	5 mos.	1 month	2 wks.	5 mos.	8 mos.	No record	3 mos.	3 mos.
Associated and etiological factors.....	Axillary lymph glands	Cervical lymph glands	Fluid in chest 5 years previously	Hæmolytic streptococcus pneumonia and Empyema, Pul. Tb.	Tbc. elbow vertebra psoas abscess. Peritonitis	Influenza 1 year previously	Addison's disease, pneumonia(?) six mos. previously	Addison's disease influenza	Pulmonary Tbc. syphilis
Side of lesion	Left	Left	Left	Left	Left	Left	Right	Right	Left
Lesion.....	Two small abscesses perforating 3rd space close to sternum	Large subpectoral abscess perforating 2nd space near sternum	Abscess anterior axillary line 7th rib tuberculous empyema	Ant. axillary line eighth *i. s.*, pulsating Empyema Necessitatis	Large subpectoral abscess perforating 2nd *i. s.*, near axilla	Large subpectoral abscess perforating 2nd *i. s.*, close to sternum. Tbc. axillary nodes	Large calcified mass fourth to seventh ribs posteriorly	Subpectoral abscess perforating third *i. s.*, to mass of calcified matter extending from third-seventh ribs in pleura	Abscess in ant. axillary line & fifth *i. s.*, X-ray shows calcified pleura
Operations....	Incision Iodine closed tight Secondary union	Partial excision iodoform closed except superficial skin drain	Excision of abscess & pieces of two ribs. Multiple rib excision	Nine aspirations. Thoracotomy with partial rib excision. Multiple rib excision. Abscess excised in multiple rib resection	Excision abscess, no drain. Exploratory lap. Resection elbow evacuation psoas abscess with closure. Drainage of psoas abscess	Excision abscess & piece of second costal cartilage superficial skin drain	None	Excision of abscess & piece of fourth rib —no drain	Excision of abscess & pieces of fourth & fifth ribs
Results abscess	Permanently closed after thirty days	Permanently closed in about three wks.	Closed after thirty-one days —remained so for 5½ mos. Small sinuses for 15 mos. occasionally closing	Pratically merged in the empyema cavity	Primary union permanently closed. Fluctuation reappeared after 7 months	Permanently closed after two weeks		Primary union, permanently closed	Recently operated on
Patient	Well	Well and in U. S. Army	In a Tb. hosp. with considerable chest wall defect. Slowly improving	Large chest defect but working and in surprisingly good condition	Died nine months after abscess excision	Well	Died	Well & working. Apparently an improved case of Addison's disease	

Texas. Weight 144 pounds. On football team; is sharp shooter. Also won blue ribbon in horse race. After coming home never was ill. No cough.

Comment: Successful result. No evidence of bone focus. Cavity behind the intercostal muscles.

CASE III.—E. F., fifty-six years, cigarmaker, Cuba; in United States twenty years.

Always a heavy smoker, a cigarmaker by trade, and generally in good health up to five years ago, when he was in the French Hospital for a week with fever, cough and pain in his chest. "One-half a gallon" of fluid was taken from his left chest, and he was told he had pleurisy.

He felt quite well after this, maintained his weight, had no cough, sweats, hæmoptysis nor other symptoms indicative of tuberculosis until one month before entering the Presbyterian Hospital in May, 1918. He then noticed a lump "the size of an egg" in the lower part of his chest in the anterior axillary line at the level of the seventh rib. It didn't seem to grow but was slightly tender and gave him dull, aching pain. At the left apex were a few subcrepitant râles and dulness with diminished breath sounds. At the left base were occasional râles, dulness and diminished breath sounds. The swelling over his seventh rib anteriorly was soft and fluctuating. The skin over it was of normal color. Pus mixed with blood was obtained on aspiration and tubercle bacilli subsequently obtained from the lesions in the inoculated guinea-pig. Sputum which was very scant showed no tubercle bacilli. A tuberculosis fixation test, it is interesting to note, was negative.

X-ray showed increase in density at the left base "suggesting fluid, and increase in density at the left apex and accentuation in the linear markings in the right upper lobe" suggesting tuberculosis.

On June 12, 1918, he was operated on for the abscess. A cavity lined with bluish red granulations and containing about 100 c.c. of necrotic tissue and grumous fluid was dissected out. No evidence of rib involvement could be made out, and it appeared that the abscess lay almost entirely superficial to the intercostal muscles. Abut three inches of the sixth and seventh ribs were removed, together with the intercostal muscles. The underlying tissues seemed healthful and the skin was closed without drainage. A compression dressing was used to obliterate the dead space.

Pathological report showed "tuberculous granulation tissue." Through a misunderstanding the wound was dressed on the seventh day. The intention was to maintain a snug pressure for twelve to fourteen days. The wound was reported clean and apparently healed. He was discharged to the Out-patient Department. Within a few days a little fluid reaccumulated. A small hole was made and some thick, bloody fluid evacuated on the thirteenth day, and pressure reapplied with each dressing.

On the thirty-first day the wound was healed and stayed closed until five and one-half months after the operation. Two tiny sinuses, discharging almost nothing, then appeared, and scabbed across and reopened for ten months. I then referred the case to Doctor Hanford, who was then concentrating his attention on the treatment of tuberculous lymph-glands and chronic tuberculous sinuses. Following a course of X-ray treatment, the sinuses closed for a few months but reopened nineteen months after operation. Twenty-one months after operation he was readmitted to the hospital under Doctor Hanford's care.

March 16, 1920, the sinuses were explored, curetted and packed with five per cent. iodoform glycerin emulsion and further radiotherapy was instituted, and he was again discharged from the hospital. Twenty-three days later, following a considerable increase in the amount of discharge for a few days, it was found that a probe be passed into a larger cavity, presumably into his pleural cavity.

SUMMARY

	1. E. M.	2. T. M.	3. E. F.	4. A. N.	5. S. W.	6. G. P. K.	7. W. L. C.	8. F. M.	9. D. M.
Age	4 1/2	16	56	19	30	23	53	37	62
Nationality ...	U. S.	U. S.	Cuba	U. S.	W. Indies	U. S.	China	Italy	U. S.
Duration of abscess	2 wks.	5 mos.	1 month	2 wks.	5 mos.	8 mos.	No record	3 mos.	3 mos.
Associated and etiological factors.....	Axillary lymph glands	Cervical lymph glands	Fluid in chest 5 years previously	Hæmolytic streptococcus pneumonia and Empyema, Pul. Tb.	Tbc. elbow vertebra psoas abscess. Peritonitis	Influenza 1 year previously	Addison's disease, pneumonia(?) six mos. previously	Addison's disease influenza	Pulmonary Tbc. syphilis
Side of lesion	Left	Left	Left	Left	Left	Left	Right	Right	Left
Lesion.....	Two small abscesses perforating 3rd space close to sternum	Large subpectoral abscess perforating 2nd space near sternum	Abscess anterior axillary line 7th rib tuberculous empyema	Ant. axillary line eighth *i. s.*, pulsating Empyema Necessitatis	Large subpectoral abscess perforating 2nd *i. s.*, near axilla	Large subpectoral abscess perforating 2nd *i. s.*, close to sternum. Tbc. axillary nodes	Large calcified mass fourth to seventh ribs posteriorly	Subpectoral abscess perforating third *i. s.*, to mass of calcified matter extending from third-seventh ribs in pleura	Abscess in ant. axillary line & fifth *i. s.*, X-ray shows calcified pleura
Operations....	Incision Iodine closed tight Secondary union	Partial excision iodoform closed except superficial skin drain	Excision of abscess & pieces of two ribs. Multiple rib excision	Nine aspirations. Thoracotomy with partial rib excision. Multiple rib excision. Abscess excised in multiple rib resection	Excision abscess, no drain. Exploratory lap. Resection elbow evacuation psoas abscess with closure. Drainage of psoas abscess	Excision abscess & piece of second costal cartilage superficial skin drain	None	Excision of abscess & piece of fourth rib —no drain	Excision of abscess & pieces of fourth & fifth ribs
Results abscess	Permanently closed after thirty days	Permanently closed in about three wks.	Closed after thirty-one days —remained so for 5½ mos. Small sinuses for 15 mos. occasionally closing	Pratically merged in the empyema cavity	Primary union permanently closed. Fluctuation reappeared after 7 months	Permanently closed after two weeks		Primary union, permanently closed	Recently operated on
Patient	Well	Well and in U. S. Army	In a Tb. hosp. with considerable chest wall defect. Slowly improving	Large chest defect but working and in surprisingly good condition	Died nine months after abscess excision	Well	Died	Well & working. Apparently an improved case of Addison's disease	

April 16, 1920, seventh and sixth ribs were removed for about 12 cm., with chest wall between.

August 2, 1920, posterior portion of seventh and sixth together with part of the fifth and fourth ribs were removed.

December 9, 1920, anterior portion of fifth and sixth ribs removed.

February 8, 1921, removal of eighth rib.

April 6, 1921, removal of posterior portion of seventh rib.

May 10, 1921, removal of anterior portion of fifth, sixth and seventh ribs.

For the past year and a half the story has been one of repeated attempts to excise tuberculous sinuses and remove tuberculous bone. He is now in a tuberculosis hospital. His condition is fair and slowly improving.

Comment: A case whose course might have been far better had he, after the first operation, had a thorough and prolonged course of anti-tuberculous treatment. This abscess did not involve bone at first and occupied the intercostal space mostly outside the muscle. An unsuccessful surgical result though not a complete failure.

Case IV.—A. N., female, nineteen years, single, United States. Cigarette packer.

Admitted to Rockefeller Institute Hospital, August 29, 1918. Had worked as a cigarette packer four years. Best weight, three months ago, 107, and has lost eleven pounds. Cough for two months.

Six days before admission had severe pain in left chest with fever, prostration but no increase in the cough she had had for two months. She had a right upper and left lower pneumonia and many tubercle bacilli were found in sputum.

She remained in the Rockefeller Hospital a little over two months, and was then referred to Otisville Tuberculosis Hospital, but refused to go. During this time, her right side cleared up, but fluid appeared on the left side that grew out hæmolytic streptococcus. She was aspirated six times: September 9, 1918, 20 c.c. yellow, cloudy fluid; September 25, 1918, 125 c.c. turbid, yellow fluid; September 27, 1918, 5 c.c. turbid, yellow fluid; September 28, 1918, 5 c.c. turbid, yellow fluid; October 5, 1918, 900 c.c. turbid, yellow fluid, one-eighth pus; October 25, 1918, 450 c.c. thicker yellow purulent fluid, one-quarter pus.

For two and one-half months she remained home, felt weak, had dyspnœa on exertion but no cough and very little fever, if any.

On January 19, 1919, she walked into the Presbyterian Hospital (Fig. 1), having noticed for two weeks a soft fluctuating swelling on left side in anterior axillary line at level of eighth and ninth ribs, that was sore. Leucocytes 12,000, polymorphonuclears 90. Slightly dyspnœic on exertion, pulse ranging from 80 to 100, temperature 99° and respirations 20 to 24. The swelling on her side pulsated with the heart beats. The remarkable feature was the disproportion between the signs in her chest and her general appearance. The whole left side was flat, with lost fremitus and breath sounds gone. The heart pulsated to the right of the sternum, and was greatly displaced. Eighteen hundred c.c. of white creamy pus was removed by aspiration and hæmolytic streptococci and largé numbers of tubercle bacilli found. She became more comfortable but began running a temperature. Seven days later 410 c.c. more fluid of the same sort was aspirated, and six days after that 1500 c.c. more. With each aspiration the abscess in the chest wall diminished in size. Her sputum, scant in amount, contained tubercle bacilli. Her temperature ran an irregularly septic course occasionally to 104°.

A serious problem was accordingly presented, namely a young woman with an enormous hæmolytic streptococcus empyema that had lasted about five months, had been aspirated repeatedly, had almost perforated spontaneously, and was associated with tuberculosis of the lungs and pleura and chest wall.

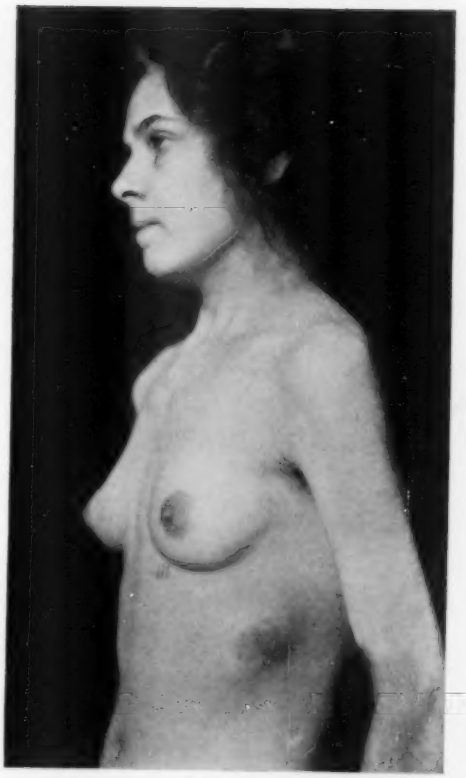

FIG. 1.—Case IV. A. N. Tuberculous abscess of chest wall due to a mixed hæmolytic streptococcus and tuberculous empyema, an "empyema necessitatis." The cavity contained over 1800 c.c. of pus. This is really a "collar button" type of abscess where the deep collection is enormous. That this chest wall abscess had its origin from the collection in the pleura is scarcely to be doubted. That other more localized pleural or mediastinal tuberculous foci may give rise similarly to the superficial abscesses seems equally reasonable.

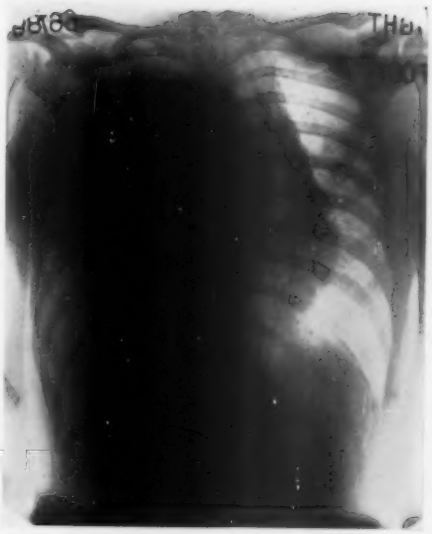

FIG. 2.—Case IV. Plate 1. Abscess with empyema necessitatis. X-ray on admission showing enormous fluid collection on left side with great heart displacement to the right. The abscess on the chest wall doesn't show. Hæmolytic streptococci and innumerable tubercle bacilli were found in the pus.

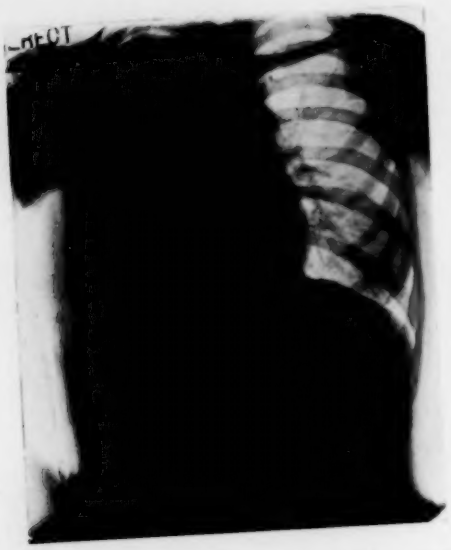

FIG. 3.—Case IV. Plate 2. Abscess with empyema necessitatis. X-ray taken the day after admission. Eighteen hundred c.c. of pus had been removed by aspiration.

FIG. 4.—Case IV. Plate 3. Abscess with empyema necessitatis. Plate taken three weeks after drainage by excision of a piece of ninth rib. In spite of suction, use of Dakin solution, etc., there is no evidence of lung expansion. The large, tubercular pyopneumothorax with the contracted tuberculous lung is shown. The appearance was just the same two and one-half months after operation.

FIG. 5.—Case IV. Plate 4. Abscess with empyema necessitatis. One month after removal of five ribs showing wide open pneumothorax with Carrel silver wired tubes (displaced from their position near the apex) in the cavity.

FIG. 6.—Case IV. Plate 5. Case of abscess with empyema necessitatis. Plate taken thirteen weeks after multiple rib resection. There is some reduction in the cavity. Four more ribs require removal, but the patient refuses to have the operation as yet.

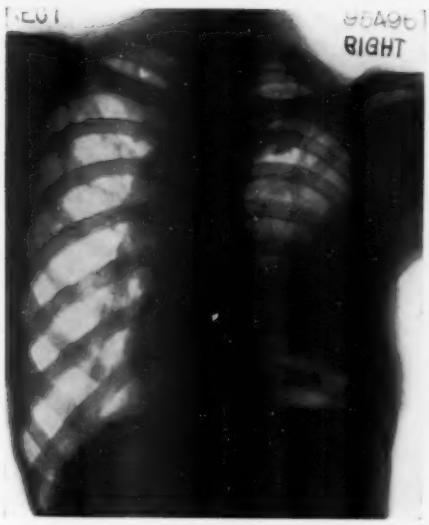

FIG. 7.—Case VII. The Chinaman who died of Addison's disease. On the right side is shown the shadow cast by the calcification and ossification of his pleural and intercostal spaces. Cf. Cases VIII and IX.

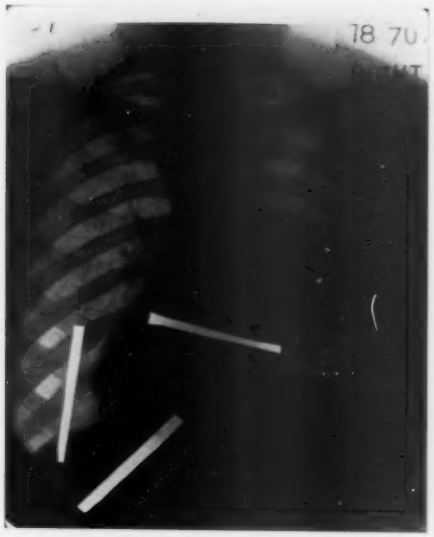

FIG. 8.—Case VIII. Plate 1. In this case many flattened calcium fragments were found and some removed. (Plate broken and repaired.) He had the symptoms of Addison's disease and improved. The calcium deposits are seen on the right side. Cf. Cases VII and IX. Taken March 27, 1920. Before operation.

FIG. 9.—Case VIII. Plate 2. Taken September 20, 1921. Operation, April 8, 1920. Shows dense calcium shadows, but less diffuse cloudiness almost 1½ years later.

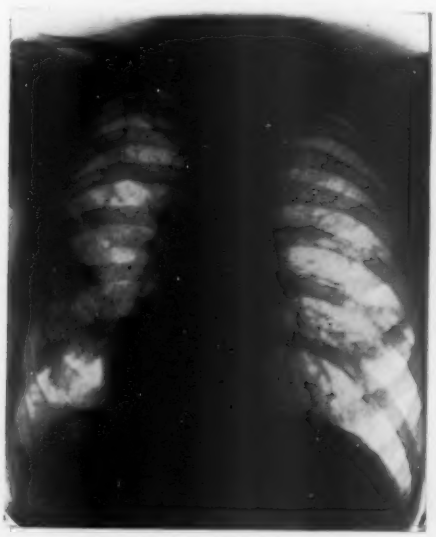

FIG. 10.—Case IX. The shadow cast by the sheet of calcium in the pleural space in the left side is clearly shown. Cf. Cases VII and VIII.

Such was the story of the tuberculous abscess of the chest wall. Its subsequent course devolved into the problem of a non-expanded tuberculous lung, even the advisability of expanding it being questionable, and a tuberculous empyema cavity. Suffice it to say that very marked general improvement followed removal of a piece of the ninth rib with drainage on February 7, 1919. Removal of most of the tenth, ninth, eighth and seventh and sixth ribs on April 24, 1919, was followed by a long period of sepsis, which, however, she finally overcame, leaving the hospital on the 294th day after admission, having regained her former weight and looking in extremely good health with a large, steadily contracting opening in her left chest wall.

She is shown as one of the cases at the meeting of the New York Surgical Society, working and in fair health, but with a sinus of considerable size that extends to the apex of her pleural cavity.

Comment: A type of tuberculous chest wall abscess, namely an empyema necessitatis, the pathogenesis of which perhaps bears a closer relation to the other cases than is generally supposed.

Case V.—S. W., male, thirty years, negro, born in West Indies.

Came to the United States ten years ago. Had always been well and working as a Pullman porter up to his twenty-eighth year, when he was sick in bed for nine weeks with "typhoid." Ever since he has had a cough and pain in elbows, especially his left. About two years before entering the hospital, he was studied in the Out-patient Department and found to have chronic pulmonary tuberculosis without bacilli in sputum but with signs of fluid in his right chest. He refused to enter the hospital at that time and visited doctors outside, and Hospital for the Ruptured and Crippled, Roosevelt and New York Orthopædic Hospital. Finally he returned and was admitted, with circumscribed, elastic and fluctuating, painless swelling beneath his left pectoralis major muscle, present for five months, swollen and stiffened left elbow for three years, swollen abdomen containing fluid and hard masses in either iliac fossæ and felt on either side of rectum, noticed for about four months, swelling and marked œdema of left leg for five months.

To condense a long and drawn-out story, he had a tuberculous abscess of chest wall in region of left second, third and fourth ribs, extending through the second interspace to a cleft between the lung and chest wall, behind the second rib, tuberculous peritonitis, with moderate amount of fluid and innumerable adhesions binding together and immobilizing distended coils of small intestine, right and left psoas abscesses, the left enormous, and before his death, about ten months later, found to contain over 1300 c.c. of pus, tuberculosis of his fifth lumbar vertebra, tuberculosis of his left elbow-joint.

In spite of it all, his general condition seemed surprisingly good though his lesions progressively increased in evidence. In retrospect, the results of the surgical attempts on his so-called surgical conditions are most interesting and instructive. They were characterized by temporary success and ultimate failure.

All of his lesions were proven tuberculosis by guinea-pig injection and actual finding of the tubercle bacilli.

June 15, 1918: The chest wall abscess was excised and its deep communication curetted out. A little iodoform left in. The wound was closed tight, dressed the fourteenth day and healed by primary union. Fluctuation reappeared in January, 1919, seven months later, but the wound never opened up to his death, nine months later.

At the same time his abdomen was opened, fluid evacuated and peritoneum washed with saline. The wound healed primarily.

He was discharged on the twenty-sixth day after his operation, July 11, 1918, to the country.

Two months later he returned to the Follow-up Clinic with his chest wound well healed and his abdomen much smaller.

Because of his improvement and because his left elbow was swollen and stiff and useless and gave him pain if he attempted to use it, he was readmitted and on August 21, 1918, elbow-joint was resected through a bayonet incision. Wound healed primarily and remained healed for three months when a small sinus appeared. It never closed.

He had been allowed to use it and had regained, at one time, about 30° flexion and extension and slight pronation and supination. It would have been better to have kept it immobilized for ankylosis.

December 11, 1918, an enormous left psoas abscess evacuated through an incision near anterior superior spine. Wall sponged off, iodoform emulsion distributed and wound closed tight in layers. Primary union. This wound remained healed until February 19, 1919, when it was reopened and over 1300 c.c. of pus evacuated.

By this time it was evident that he was so overwhelmed by tubercle bacilli that further operating should be for symptoms giving him pain. He was having diarrhœa and obstruction symptoms. The psoas abscess was accordingly opened and allowed to drain. Whereas he had been running an irregularly septic temperature, this dropped to a normal range with subnormal excursions and he died two months later on April 20, 1919.

Comment: The chest wall abscess showed no rib involvement that could be found. It never reopened after excision with curettage of the deeper pocket beneath the intercostal muscles, though fluctuation returned over half a year later, in spite of the fact that the patient was practically riddled with tuberculosis. The elbow resection was probably poor judgment to attempt at the time it was done. Continuation of his anti-tuberculous régime at that particular time would have been better.

Case VI.—G. K., female, twenty-three years, United States.

At six years tonsils and adenoids removed. At fourteen years measles. At eighteen years began work as stenographer. At twenty-one years influenza. Sick two weeks. Weight 110 with clothes on. At twenty-one operation for adhesion about cæcum and chronic appendicitis. At twenty-second year, September, 1919, first noticed a six-cm. lump upper inner quadrant of left breast.

Soon afterwards felt a painful lump in left axilla. Was advised that she had a tumor in her breast that would require breast removal. For the past year not as vigorous as usual.

April 27, 1920: First examined by writer. She was then twenty-three years old; temperature 99.8°. Slender young woman, weighing 101 and evidently under weight. Lungs clear, no râles; cogwheel breathing left base posteriorly and at sides. A negative physical examination otherwise, except for a diffuse fluctuating swelling about 15 cm. across in region of the second and third left costal cartilages; slightly movable on chest wall with pectoralis major slack, but quite fixed when contracted. It was evidently beneath the breast and pectoral muscles. A rather large mass, over 4 cm., was readily felt in the left axilla. X-ray report by Dr. A. H. Busby "shows no definite evidence of pathology in the bone substance of the anterior upper left ribs, but there is a small peculiar shadow showing abnormal density over the situation of the cartilage in the anterior portion of the upper left second rib, which makes me suspicious of some pathology in the cartilage at this point. Whether the process is actually in the cartilage or due to a soft tissue opacity in the upper portion of the left breast it is impossible to differentiate, yet I am suspicious that it is cartilage at fault. The heart and vessels are negative. There is some peribronchial thickening which is slightly more extensive in the left lung, numerous enlarged glands are visible about the

roots of the lungs and a few calcified nodes are seen. Both apices show a slight infiltration, more in the right than the left, which is suspicious of a tuberculous infection. It is of course very slight in extent, yet the right apex suggests activity."

Operation, May 11, 1920: Pathology—The axillary mass consisted of caseating tuberculous lymphnodes. The main abscess cavity was beneath the pectoral muscles. The cavity was about 12 cm. in diameter. It was lined by thick bluish-red tuberculous granulations, and contained much turbid fluid with flakes and masses of coagulation necrosis matter in it. Between the 2nd and 3rd costal cartilages ran a small sinus to a deep pocket between lung and thoracic wall, running upwards behind the cartilage of the 2nd rib containing about an ounce of thick, coagulation necrosis material.

The costal cartilage was a little uneven but no rib necrosis nor evidence that rib or costal cartilage were primary foci could be determined.

Procedure: An inframammary incision carried toward axilla was done. Axillary nodes removed. Breast reflected upward on neck and pectoralis major split longitudinally and as much of abscess enucleated as possible before breaking into it. Sinus carefully curetted, a little iodoform powder dusted into it, muscle closed by suture and skin closed with two little rubber tubes just through skin at either end of incision. A snug pressure bandage applied to as far as possible obliterate dead space.

Pathological Report—by Dr. A. P. Stout. Tuberculosis of abscess wall and axillary nodes. The cartilage, "on cut section it shows no gross abnormality." "The costal cartilage showed a very marked irregularity in the staining process and there is irregularity in the arrangement of the cells."

Culture of fluid from abscess—"sterile." M. Mueller. Guinea-pig injection. "Smears show presence of tubercle bacilli." R. H. Pauli, May 12, 1920. Autopsy, June 24, 1920. "Tubercles in spleen, liver and lungs." "Microscopic positive for tuberculosis." W. C. Von Glahn.

Sutures and drains out on sixth day, discharged on thirteenth day. Scabs over drainage tract. In good condition and afebrile. Weight 99 pounds.

Follow-up note: Four months, Setember 7. Weight 107¾ pounds, more than she had ever weighed in her life. Taking cod-liver oil and is sunburned. Feels well. Scar healed and has never broken down. No evidence of recurrence of lump. Six and one-half months, November 22nd. Weight 112½ pounds. Feels very well indeed. November 23rd, examined by Dr. A. R. Lamb. Temperature, 98.6°; pulse, 100 (excited); blood-pressure, 135-90. No tuberculosis symptoms; at top weight and better than ever before in her life. Chest—good expansion, including apices and bases. Absolutely normal all over except slight catchy respiration over left lower lobe. Returned to work.

One year—May 3, 1921. Weight, 107¾ pounds; temperature, 99.2°; pulse, 90; respirations, 18; blood-pressure, 115. Lungs clear. Wound O. K. No recurrence of swelling. Somewhat tired after winter's work, but feels quite well otherwise. July 15, well 103 pounds. October 26, 1921, one year five months. Presented at New York Surgical Society meeting. Feels quite well; 112 pounds. No recurrence of abscess.

Comment: No rib lesion demonstrable. Abscess communicated with a cavity beneath the intercostal muscles. A case treated under favorable anti-tuberculosis conditions.

Case VII.—W. L. C., male, fifty-three, cook, Chinaman—S., four years in United States. No history of previous respiratory trouble, but was sick in bed with fever in California several years ago. Loss of twenty-nine pounds in six months. Six months ago caught cold, chills, and four or five days fever. Ached in shoulders and hips. Soon noticed dyspnœa on exertion; weakness, and had to stop work;

darkening of lips and gums for four months; darkening of skin, gradual and progressive; sore mouth; anorexia and slight abdominal pain; night-sweats a few months ago; nocturia three to four; emaciation. Skin dark brown with darker blotches, more on body and arms than on legs. Tongue and gums show irregular patches of blue black color. Slight dorsal scoliosis to left. Left chest larger than right and showed compensatory emphysema. Right chest immobile, dulness, fremitus absent, diminished breathing, diminished voice. No râles. Blood-pressure 86-56. Arteries soft. Right upper quadrant tender and resistant. Blood count, 5,000,000; white blood-cells, 9600; polymorphonuclears, 58; hæmoglobin, 75 per cent. Wassermann negative. X-ray, September 29, 1921. Definite dense shadow in lower part of right lung. Blood urea, 0.9 grams per l. Creatinin, 1.5 mg. per 100 c.c. Gastric—free HCl, 30; total acidity, 54; guaiac negative. No fever on admission.

Died rather suddenly early one morning four days after entering hospital, after a chill and elevation of temperature to 103.4°.

Clinical Diagnosis: Addison's disease. Calcification of pleura.

Comments: Though this case had no actual abscess, his chest wall was so similar in other respects to Cases VIII and IX that he is included in this series.

AUTOPSY REPORT ON CHEST WALL

"*Chest Wall.*—On the right side, beginning with the fourth rib and extending down to the tenth rib, there is extreme thickening of the chest wall, with calcification of the intercostal muscles. The thickness of the chest wall through this region is perhaps 3 to 4 cm. Near the vertebral column, over the sixth, seventh, eighth and ninth ribs, there is a depressed crater, about 3 cm. in circumference. This probably represents an old encapsulated abscess. The tissue about this crater is densely calcified; the floor covered by a continuation of the smooth, opaque, thickened pleura.

"The right lateral chest wall of the thorax from the fourth to the tenth ribs was removed. After fixation, a vertical saw cut was made and the following condition noted: The ribs themselves contain a normal red marrow throughout their whole length and are not abnormal. The thickening of the thoracic wall is found to be due to a bony plate, consisting of an inner and outer table, separated by red and yellow marrow. The outer table is about 3 mm. in thickness, the inner one a little thinner. This bony plate is covered on the pleural surface by a glistening, white, opaque, fibrous membrane, to which shreds of lung tissue are still adherent. The lesion is thus confined to the pleura itself."

Beyond a small superficial scar at the left apex the lungs were rather strikingly free from the lesions of tuberculosis.

Most of both adrenals were replaced by tubercle tissue. The lymphnodes about the pancreas showed tuberculosis.

CASE VIII.—F. M., male, aged thirty-seven, Italian; railroad laborer. Measles in childhood and "malaria" at fifteen. Came to United States from Italy when eighteen. Married at twenty-one; wife is well and has had eight pregnancies, but three children died. She had no miscarriages. Remaining children well. Worked as laborer drilling rock up to ten years ago. Since then a track laborer on railroad. At thirty-six years, in 1919, one year before admission, weighed 149 pounds, which was as much as he had ever weighed in his life.

In May, 1919, ten months before admission, was in bed fifteen days with "influenza." He had an eight-day cough with sputum that was not bloody nor yellow. Was "laid up" for one month. From this time on he has not been well. He returned to work throughout the summer and fall for six months. He dates the appearance and increase of pigmentation of his face and other portions

of his body from this time. For three months he has noted a lump appear and grow to its present size just above and inside his right nipple region. Finally, a month ago, pain began after lifting heavy weight that he had been accustomed to frequently lift before. Two weeks ago this became severe enough to stop his working. The right shoulder and back of his neck gave him most trouble, especially at night and on moving his arm.

His weight is 117, a loss of thirty-two pounds in a year. He is steadily growing weaker and complains of gas eructations following meals.

Physical Examination: The most conspicuous thing in his general appearance was the pigmentation of his face, flexor surfaces of his arms, shoulders, waist line and where pressure points exist. One very small pigmented spot was noted on the buccal mucous membrane of cheek.

A few discrete enlarged nodes in right axilla, fewer in neck and one in left axilla.

On anterior surface of right chest with fourth rib as a centre just outside the costochondral junction and over the third and fourth interspaces, a soft fluctuant swelling about 10 cm. in diameter. It was beneath the pectoral muscle and but slightly tender. Expansion of right chest is diminished. There is almost flatness and much diminished breath and voice sounds at right base anteriorly and laterally. Fremitus is lessened.

His heart was negative, his arteries soft and his blood-pressure low. Weight 117.

Blood-pressure: March 24th 94/78, March 29th 88/70, March 30th 82/70, March 31st 82/70, April 1st 86/70, April 2nd 90/68, April 3rd 94/72, April 6th 88/66, April 8th operation, May 7th 80/60, May 15th 78/40, May 16th discharged.

Blood-pressure (follow-up): June 7th 90/50, weight 132; September 1st, weight 125; September 3rd, 75/50, weight 128½; December 13th, weight 149½; December 20th 115/75, weight 150; (1921) January 17th, weight 148; January 31st, weight 151¼, June 27th 115/80, weight 144; July 12th 90/65, weight 143; September 20th 110/78, weight 145½.

Blood chemistry, basal metabolism, phthalein and gastric test meals were all normal in range. The red blood-cells, 6,000,000; hæmoglobin, 80 per cent.; leucocytes, 8000, and polymorphonuclears 70 per cent. Von Pirquet was positive. Wassermann negative.

X-ray showed "large quantity of calcified material in the right side of the chest, which extends from the third to the seventh rib anteriorly and seems to be due to calcification of the pleura."

A week before operation following the slight exertion incidental to metabolism test, patient became prostrated and complained of pain in back of neck and abdominal cramps. It looked as though at that time he might die.

Early the next morning he had severer pain in his back and vomiting. During the stay in the hospital, and even after discharge, for several months various observers commented on the increase in the density of the pigmentation.

For some reason or other a more than usual interest was taken by the various members of the interne and attending staffs in the diagnosis.

Because of the asthenia, pigmentation, low blood-pressure, gastro-intestinal symptoms and the lesion in his chest, when it was found to be tuberculous, the consensus of the whole staff was that he presented the symptoms of Addison's disease and his prognosis altogether bad.

At various times during the study of the case the chest wall lump was considered empyema necessitatis, gumma, tuberculous abscess, lung tumor, hypernephroma metastasis, simple abscess of rib, and it was rather interesting that so many views should have been advanced.

Aspiration was done and 10 c.c. of greenish-yellow fluid sterile in aërobic and anaërobic media was obtained. It contained 75 per cent. polymorphonuclear cells. Tubercle bacilli were subsequently recovered at the autopsy of inoculated guinea-pig.

Operation April 8, 1920: One per cent. novocaine; last part, gas and oxygen.

Pathology: Beneath pectoralis major lay an abscess full of about 60 c.c. of greenish, creamy pus with flakes of necrotic granulations throughout. Wall was sharply circumscribed. In third interspace, running downward and outward to pleural space behind the fourth rib and fourth interspace, and even behind the fifth rib, was a crevice or slit, lenticular in shape, full of fragments and plaques of calcium salts. Some of these were loose and readily removed, others not.

Pectoralis major split, abscess wall dissected out. Portion of fourth rib excised. Many fragments and plaques of calcium removed and cavity smeared thinly with a little iodoform suspended in oil. Pectoralis minor muscle origin from rib cut away and muscle packed into sinus. Pectoralis major sutured and skin closed tight with no drain whatever. Pressure dressing applied.

Forty-eight hours postoperative; temperature to 106.4°; and iodine was found in urine. Within two days temperature came to normal and remained there.

Wound dressed sixth day because it was getting a little loose. Stitches removed. No sinus. Wound healed by primary union and never opened. Transferred to medical wards on eighteenth day, where he was given a high caloric diet.

Discharged thirty-eighth day, postoperative.

He has been seen twelve times in follow-up clinic and is now a wholly different picture.

He has gained twenty-five to thirty pounds in weight.

Pigmentation has disappeared.

Strength is fast returning, and the wound has remained healed with no recurrence of abscess though his calcium plaques are still present. At his base is dulness, diminished breathing and a few râles.

His blood-pressure has returned to a low normal. (See blood-pressure table above.)

Comment: He is considered by all who have seen him as an improved case of Addison's disease; from this standpoint alone, a case worthy of note and careful following. No rib involvement was found. It seems likely that he had a flare up and possibly pleural exudate at the time he had "influenza," but it is hard to believe so much calcium could have been formed in so short a time—ten months. It seemed likely that he had tuberculosis in his lung or pleura or in his mediastinal lymphatics much longer. Case shown before the New York Surgical Society, October 6, 1921.

Case IX.—D. M., male, sixty-two, carpenter, United States.

At twenty-five, thirty-seven years ago, Neisser infection. No history of lues. At thirty-three, twenty-nine years ago, pneumonia, but a certificate made out by his doctor for insurance benefit was signed "phthisis." It took him "one year to cure this" by an out-of-door life and creosote internally and by inhalation. At fifty-eight, in 1918, he had influenza with a bad cough for three months. Went to work at Long Beach and in three weeks his cough stopped. Six months ago had a temporary recurrence of cough. Three months ago soreness developed in his left side and two months ago found a small lump over region of fifth rib in anterior axillary line.

A thin, wiry man of 124 pounds, who looks as though he had lost weight. There are subcrepitant râles at both apices and in first interspace on left side. There are occasional râles at both bases. Breath sounds, voice, fremitus and resonance are not very markedly changed. In the anterior axillary line over the

fifth rib is a fluctuating, slightly tender swelling not attached to skin, not pulsating, and fixed on chest wall.

X-ray shows an irregular mottled area of shadow suggestive of calcification of pleura. Red blood-cells, 4,200,000; hæmoglobin, 80 per cent.; leucocytes, 8200; polymorphonuclears, 75 per cent. Blood urea, uric acid and sugar are normal. Urine is quite negative. Blood—Wassermann: Chol. four plus; alc. four plus.

Aspiration of abscess: Grayish, caseous-appearing material containing much cellular débris. No organisms seen.

Case shown before operation at the New York Surgical Society, October 26, 1921.

Subsequent Note: Operation, October 29, 1921. Local anæsthesia. Abscess excised and about 12 cm. of fourth and fifth ribs removed with intercostal muscles. The upper margin of the fifth rib was slightly concave, as though there had been some decalcification, but this seemed a small lesion compared to that on either side of it. The pleura had been changed to a firm sheet of smooth, calcified, possibly ossified material that could be tapped on with an instrument, producing a resonant tympanitic sound, due to the subjacent lung. A sinus from the abscess cavity only about 0.5 cm. in diameter led to the surface of this sheet of calcium at the level of the fifth rib and was attached to a very small punctate depression that would not admit the end of a probe.

The wound was closed after smearing in a small amount of iodoform and splitting the pectoralis major so as to draw it across the sheet of calcium, suturing it to the serratus magnus.

Tubercle bacilli were found in the pus from the abscess. Twelve days later he was doing well.

OBSERVATIONS ON A CASE OF POST-OPERATIVE TETANY WITH IMPLANTATION OF HUMAN PARATHYROIDS

By Arthur E. Brown, M.B. B.C., Cantab.
of Colac, Victoria, Australia

In the Annals of Surgery for February, 1911, Dr. W. H. Brown of Colac published the details of a very striking case of cure of post-operative tetany by transplantation of human parathyroids. The case I report now was also originally under his care and was treated by me in conjunction with him.

The patient, a woman aged thirty had had the right lobe of the thyroid removed eight years ago, in a neighboring city. While in bed recovering from the operation she had an attack of cramps, which had ever since recurred with great frequency. The longest interval between them might be two to three months, but they were decidedly most severe at about the onset of the menstrual periods. Eighteen months before she had become pregnant and had been entirely free from attacks throughout pregnancy: After her confinement, however, they recurred with greater frequency and severity and rendered her unable to do her usual housework. She was a stout woman and her general mental state was one of intense neurosis. She was absorbed in herself and her illness and described "queer feelings" to an unlimited extent. Her attacks consisted of cramps in the limbs and body with severe pains round the heart. The hands assumed the typical tetany or "obstetric" position. The elbows were strongly semi-flexed, the ankles plantar-flexed and the toes pointed. Her pulse in the attacks was from 100-110 and she was afflicted with severe terror. Chvostek's facial nerve sign was present.

She was in this condition when first seen on August 21, 1919. She was given thyroid sicc: gr. ii with calcium glycerophosphate gr. xx daily. A month later she reported that she had a good deal of stiffness with profuse sweating. The calcium dose was increased up to thirty grains thrice daily. Larger doses than this made her sick and were refused. Her attacks were relieved to a large extent by one drachm doses of syrup of chloral.

On December 8th a woman of fifty died in the hospital of exophthalmic goiter and three parathyroids were obtained from her body and implanted within an hour, under the aponeurosis of the external oblique muscle. Some difficulty was experienced in freeing the graft bed from fat. The wound in the succeeding days showed signs of breaking down but no actual suppuration occurred. There was no improvement, even temporarily, from this operation. For the next four months her attacks were frequent and severe, one every forty-eight hours or less. Her mental state was extremely neurotic, and her pulse varied from 80 to 100. Her condition in April, 1920, was worse generally, both cramps and neurotic symptoms being very marked. Her temperature during the month rose gradually to a maximum of 101° Fahrenheit, and fell as gradually to normal. No intercurrent disease could be discovered.

On April 16th one bullock's parathyroid was obtained from the abattoir, and injected emulsified in glycerin intramuscularly. No obvious effect was produced. Her condition got generally worse until May 25th when four more bullock's parathyroids were secured and injected. This gave her immediate relief, continued for a week, at the end of which time the cramps and nervous symptoms began again to increase. Four more bullock's parathyroids were injected on June 8th, but

on this date also I was able to secure three human parathyroids from the body of a boy of seventeen who died from the results of an accident. These were implanted within forty minutes of his death, inside the sheath of the left sternomastoid muscle. No fat was encountered at this operation, and the sheath was sewed over the glands in a clean graft bed in the muscle. Chloroform anæsthesia was used, and the only noticeable occurrence was that during the induction of anæsthesia the limbs assumed the typical tetany position. The wound healed well by first intention.

On June 19th and 20th she had two short "fits," the characteristics of which were tonic spasm of the muscles without the tetany position, tachycardia, and a short period of unconsciousness, absence of terror and amnesia as to the fit. She was taking calcium lactate and glycerophosphate, fifteen grains of each, at night only, and though her nervous symptoms were still bad, she had no cramps since the operation. A week later her mental condition was very much better. She was for the first time in my acquaintance with her taking an interest in people round her and spoke much less of her "queer feelings." She was discharged from the hospital with instructions to take one of the calcium powders only when she felt it necessary.

On July 15th she reported herself as very well, going out for walks and sleeping well. Two months later she came to see me and reported herself as very well indeed and able to work in the house all day for the first time since her confinement, eating and sleeping well. Mentally her outlook appeared to be very much better, and she had not taken any calcium for three weeks. Chvostek's sign was still present. Three days before her August period she had a slight stiffness in the hands while out walking. The menstruation in September, was normal in every way.

On November 5th she came in again in a very bad condition. She had diarrhœa for three days. Her temperature was 101° Fahrenheit, pulse 120, respirations 60, not obstructed. She cyanosed and very terrified. The hands and arms were clenched, but not in the tetany position—the toes pointed and the calves stiff. Nothing abnormal was discovered in the lungs, the heart apex was external to the nipple line. There was no œdema. The urine contained a small quantity of albumen, no acetone or other abnormal constituent. Syrup of chloral was given without effect. Parke Davis's parathyroid extract was given intramuscularly in 1/10 grain, repeated in two hours without any result. During the next two weeks her condition gradually improved, the respirations getting quite easy, 30 to the minute and the pulse 98. There was no further sign of muscle stiffness since the first night she came in. Some dullness and moist sounds appeared at the base of the lungs. She was very hysterical shrieked with terror if the room door was shut, globus hystericus was present intermittently, and other varying symptoms including blindness. She was absolutely unable to sleep and narcotics had no effect whatever. By December 11th considerable œdema developed in the back and legs. Bullock's parathyroids were injected intramuscularly without any improvement. She began to respond a little to paraldehyde and on the 12th of December I was able to induce a light hypnotic condition in which her normal voice, vision and respirations were reproduced, but relapsed at once on awakening. Two days later her pulse was 80, respirations 30, temperature 98.4° Fahrenheit. The albumen was less and she was inclined to sleep. The nurse had left her bed for a few moments and returning found her dead.

On post-mortem examination, the body was intensely cyanosed, the veins everywhere being congested with blood. The abdominal cavity contained a very little clear fluid. The alimentary canal was normal throughout. The liver weighed 52 ounces and was normal in consistence with some nutmeg appearance on section. The spleen was somewhat enlarged and congested. The kidneys stripped easily of their capsules and showed some thickening of the cortex. The

pancreas was enlarged and very hard. The lungs were soft, not consolidated or greatly œdematous, although they partook of the general congestion. The heart was moderately enlarged and contained post-mortem clot only. The valve orifices were of normal size. The larynx was removed with the thyroid in situ. There was no evidence of œdema or obstruction in the glottis. The thyroid isthmus was enlarged and hard, but not obstructing respiration. The ovaries, pancreas, suprarenals and thyroid were sent to Dr. S. W. Patterson at the Melbourne Hospital for microscopical report. He reports "the right lobe of the thyroid has been removed practically entirely at the operation and the isthmus is now considerably hypertrophied. The thyroid gland in section shows no obvious histological abnormality however. I was unable to demonstrate any parathyroids remaining attached to the neighborhood of the thyroid after cutting sections of many likely portions. The pancreas showed increase of fibrous stroma, more noticeable in sections from the tail of the gland, in which part also the parenchymal cells were somewhat degenerated, staining diffusely with eosin. There is no evidence of small-celled infiltration in the connective tissue. The islands of Langerhans appear normal throughout the gland, but the blood-vessels in all portions were decidedly thickened. Sections of the ovaries and suprarenals present normal appearances."

He examined also for me sections of the sternomastoid muscle at the site of the engrafted glands, and reports: "In the scar of the sternomastoid I found remnants of three small glands which although degenerated have the appearance histologically of parathyroid tissue." His opinion expressed to me was that these glands were probably not functioning at the time of death. A microphotograph of one of them is shown.

The case presents several features of interest, such as the lengthy duration of the condition; its association with the menstrual periods, pregnancy and lactation; the reaction to the various forms of treatment, and the anomalous nature of the relapse which led to her death.

The length of the trouble is unusual in these cases. The trauma was evidently sudden; the damage evidently done at the time of the operation for her attacks came on within a few days of it. Apparently a condition of partial parathyreopriva was produced, the amount of gland substance remaining being sufficient to maintain her in health for six or seven years during which time her attacks did not increase in frequency or severity. It does not seem to have been sufficient to stand the stress of times of glandular strain, such as menstruation, efficiently; and apparently broke down altogether under the severe strain of lactation, for her condition continued to deteriorate after she had weaned her baby. What the exact relationship between the ovarian, parathyroid and other gland functions is, is of course still obscure; but the fact that menstruation, pregnancy and lactation do throw a strain on parathyroid metabolism is well known experimentally. Alquier and Theunveny[1] found that in dogs after partial parathyroidectomy, the menstrual periods were less frequent, and of briefer duration, and pregnancy was more difficult to obtain. Ochsner and Thompson quote a case reported by Pineles in which the operation was performed during pregnancy. Tetany followed in four days, lasted fourteen and recurred with a subsequent pregnancy. The same authors in their book report experiments on animals after parathyroidectomy, showing tetany occurring in sharp attacks during pregnancy and lactation.

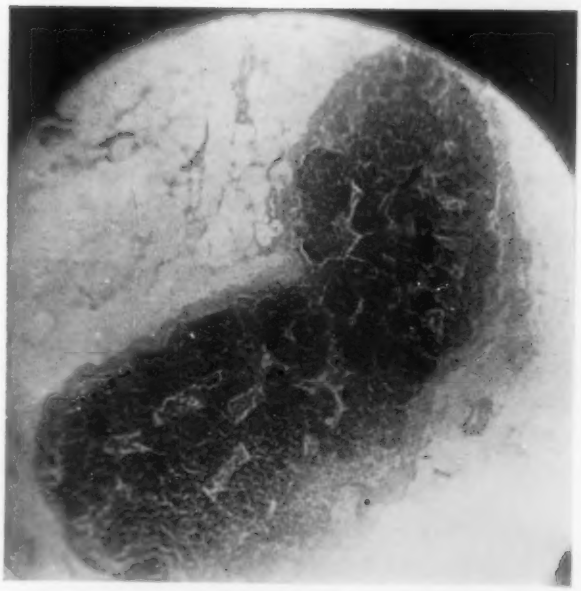

FIG. 1.—Microphotograph of parathyroid graft in fibrous tissue from sternomastoid muscle.

The theory of McCallum claims that these results are due to the loss of calcium suffered by the maternal body in building up the fœtus and in producing milk, thus throwing strain on a calcium metabolism already disordered by parathyroid deficiency. It is interesting to note that in the present case, although the menstrual and lactational relationship to tetany correspond with this experimental evidence, the patient was entirely free from attacks of tetany during her pregnancy. In regard to the controversy as to tetany production a recent worker[2] has published the opinion that calcium deficiency has little to do with the production of parathyroid tetany. He regards methyl cyanamide as the mother substance of guanidines which are responsible for the toxic symptoms and recommends acid therapy regulated to just below the point of acidosis. Shepherd of Montreal[3] claims to have succeeded in a permanent cure of a case by large doses of calcium lactate (one drachm every three hours). His case was discharged taking half a drachm of calcium lactate with 1-20 grain of parathyroid extract four hourly. He criticizes W. H. Brown's treatment of his previous case on the ground that the calcium was given in too small doses. We have found that larger doses cause the patient much discomfort and sickness; and Shepherd states that he had difficulty in persuading his patient to continue the treatment owing to its unpleasantness. In any case medical treatment in such a case as the present one would have to be indefinitely prolonged; for the history of the case indicates that the remaining gland substance is not capable of taking up sufficient burden to compensate for the lost tissue. Parathyroid extract by the mouth has failed wherever it has been tried; but seeing that fresh parathyroid has been repeatedly reported as ineffective when given by mouth, whereas we found it to confer very great relief on intramuscular injection, one wonders if the active principle is destroyed in absorption from the alimentary canal, and if a soluble preparation for intramuscular injection would not be a more hopeful procedure.

As to the transplantation of parathyroids, the difficulty is of course to get the opportunity of taking the glands from a human subject. W. H. Brown's experience with parathyroids from animals goes to show that these glands do not survive as grafts in the human body. Eiselsberg [4] and Stenvers [5] have reported cases in which parathyroids were obtained during operations for thyroidectomy on other patients and transplanted with successful results. But one would hardly consider such a procedure justified, knowing the great variability of the glandules in size, number and position; and more particularly in the face of such a case as this where tetany supervened after the removal of one lobe of the thyroid only. We considered for some time the possibility of obtaining parathyroids from stillborn infants. According to Erdheim, however, the functionating cells are first found in the parathyroids at about the tenth year of life; and Forsyth states that only in one case has colloid substance been found as early as three months[1]; so that it is not likely that this method offers any real hope of success.

The first attempt to graft failed. The glandules were obtained from an exophthalmic case aged fifty. The experience of Eiselsberg and Stenvers

shows that there is no inherent reason against using the parathyroids of exophthalmic patients. And Ochsner and Thompson state that "the parathyroid glandules are in no way associated with the thyroid, save for the relationship of anatomical propinquity, and that functional relationship (which may in certain instances be more intimate than we suppose) which must exist between all important glands that have to do with internal secretion." I think the first graft failed owing to the difficulty of freeing the graft bed from the fat that was so abundantly present. For the second attempt I had no difficulty in securing a good graft bed free from fat. Why, after causing such immediate and complete recovery lasting for five months, they should have ceased to function and have degenerated I do not know. That such grafts do live permanently the cases quoted above prove. The woman reported by W. H. Brown in 1911 is still a normal healthy woman after the lapse of ten years. The relapse was particularly striking in that it showed no features which apart from her history would lead one to suspect tetany as the cause. Her transient stiffness on her readmission was certainly not sufficiently characteristic to arouse one's suspicions. Chvostek's sign persisted throughout, however, and was the only sign remaining when her mental and physical disabilities had completely disappeared to make one anxious about the permanence of her cure. The way in which her mental symptoms cleared away after the second implantation of glands was most striking and brought the mental aspect of the disease into strong relief; which was unfortunately still more strongly emphasized in the relapse which led to her death.

I have not in this paper reviewed the evidence on which rests the belief that the parathyroid glands are separate in function from the thyroid, and that it is by their destruction that tetany is produced, believing that this view is now the generally accepted one. In the British Journal of Surgery for April, 1921, however, so great an authority on thyroid work as Mr. James Berry of London declares his frank scepticism as to the separate functioning of the parathyroids.

He states that he has done 1338 thyroidectomies without troubling about the parathyroids, only leaving a small piece of gland behind at the hilus; and he has had no tetany occurring early or late. He considers tetany would only occur in total thyroidectomy because no thyroid substance was left. It was a similar opinion expressed by some men of great judgment and experience in the journals about that time that led W. H. Brown into disregarding the parathyroids in a difficult case in 1910; with the prompt occurrence of tetany as a result.

This case is as far as I can ascertain the only one published in which tetany has supervened on the simple removal of a single lobe.

REFERENCES

[1] Ochsner and Thompson: Thyroid and Parathyroid Glands, 1910.
[2] W. F. Koch: Med. and Surg., 1918, vol. ii, p. 9.
[3] Annals of Surgery, Nov., 1912.
[4] Quoted by Ochsner and Thompson.
[5] Quoted by Lancet, April 21, 1917.

LETHAL FACTORS IN ACUTE ILEUS*

By Frederick T. van Beuren, Jr., M.D.
of New York, N. Y.

Why do people die of acute intestinal obstruction? What are the factors responsible for the appearance of the symptom-complex of acute ileus and which of them are essentially fatal? These are questions which occur to anyone who reviews a series of such cases. These are questions which must be answered, provisionally at least, if we are seriously to attempt any improvement in the present mortality rate: a rate which can be favorably compared only with those of such surgical catastrophes as thrombosis of the pulmonary artery, neglected gas gangrene and major battle casualties.

But, to make our provisional answers intelligible—even as a basis for controversy—we have first to define the conceptions of intestinal obstruction and of acute ileus which we have used to argue from. For this purpose—

(1) Acute intestinal obstruction may be defined as a local condition associated with the sudden abnormal stoppage—from any cause—of the intestinal current between stomach and anus.

(2) Acute ileus may be defined as a general condition whose symptom-complex appears when acute intestinal obstruction—from any cause—has existed long enough to make its effects manifest.

There is a very practical reason for emphasizing this differentiation and I make haste to state my belief in it lest I be charged with meticulous precision. The proper treatment for acute ileus, whatever its underlying cause, is similar in every case; while the appropriate treatment for intestinal obstruction varies according to its mechanical, spastic or paralytic character. Whoever fails to appreciate the practical difference between obstruction and ileus may readily fail to recall that the latter deserves its proper treatment quite as much as the former. And, again, if he fails to apply his knowledge of this fact he will lose cases of acute ileus that he ought to save.

Acute intestinal obstruction bears to acute ileus roughly the same relation that a wound contamination bears to a systemic infection. One is a local condition and an underlying cause of the other. The other is a general condition, dependent upon and arising from the local lesion. But the general condition appears only when its local cause is active to a sufficient degree and for a sufficient time.

We readily differentiate between a healthy carrier of the bacillus and a person suffering from diphtheria. Why not differentiate, then, between a case of acute ileus and its underlying local condition of intestinal obstruction?

* Read before the Section on Surgery of the New York Academy of Medicine, April, 1921.

† Cases and percentage cited are from human series at the Roosevelt Hospital and from animal series at the Laboratories of the Department of Surgery, Columbia University.

And though one may feel that I have unwarrantably prolonged two lines of argument that are perhaps not parallel, I feel that—for practical purposes of illustration and emphasis—I am justified in doing so.

To develop this conception a little further may I remind you that we do see cases in which we make a tentative diagnosis of intestinal obstruction but in which the symptoms spontaneously disappear in so short a time that operation is not done. An illustration of this is the very recently irreducible hernia with local pain and tenderness and reflex vomiting of which the house surgeon has notified you and which reduces spontaneously while you are on your way to the hospital. There is no ileus symptom-complex apparent in such a patient; even though the local condition persists for several hours and requires operation for its relief. No ileus condition exists and no treatment for it is required. On the other hand, we see cases of hernia which have remained in a condition of unrecognized obstruction or strangulation for an entire day or two days or even longer. They, too, show a tender, painful, local swelling where the bowel is obstructed, but they show other signs in addition. In such cases the symptom-complex of ileus is apparent, for there have been causes active and there has been time allowed for the development of intestinal damage, for poison formation in the obstructed intestine and for general tissue dehydration. In such cases the ileus condition does exist in addition to the obstruction and it does require treatment just as much as the strangulated hernia.

The point I make has quite a definite application to the series of cases a part of whose analysis I desire to offer in evidence, for I have included in the series all the cases of obstructed or strangulated hernia together with the cases of intestinal obstruction from all other causes that have been operated for acute ileus at a large city hospital during the past ten years. I have included them in spite of my appreciation that they are frequently omitted from such a series. I have included them because I wish to emphasize my belief that—in the interest of better mortality statistics for strangulated hernia as well as for other mechanisms of the underlying cause of acute ileus—they should be so included. I have included them in order to make the point that acute ileus resulting from strangulated hernia deserves treatment quite as much as acute ileus arising from the common post-operative mechanisms such as bands and adhesions.

The point is this: ileus is ileus whatever mechanism be the underlying cause (and I use this term mechanism in its broadest sense). Both the ileus and the underlying cause deserve prompt treatment.

The facts are these: In strangulated hernia ileus, the hernia is the prominent feature. It receives prompt treatment and the ileus does not. In ileus that arises from other causes than external hernia, the ileus is the prominent feature and neither the ileus nor the underlying causes receive prompt treatment.

Evidence in support of this statement can be found in the mortality statistics of intestinal obstruction from almost any general hospital. The

death-rate varies from thirty odd to sixty odd per cent. and averages between forty and fifty per cent. Now our own operative statistics show that the mortality is less than twenty per cent. where operation follows within twenty-four hours of the onset of symptoms, and over seventy per cent. where operation is delayed for over seventy-two hours. Even at the end of forty-eight hours after onset, operation gives only a little better than a fifty per cent. chance of recovery. Here then is two-way evidence that prompt treatment is needed and that it is not often received. Only thirty-three per cent. of our cases in whom the elapsed time could be satisfactorily calculated were operated within twenty-four hours after onset of symptoms.

Of course it must not be thought that this was entirely our fault. As matter of fact only twelve per cent. of the case series originated in the hospital: the rest applied from outside and were mainly "emergency operations" done as soon as the attending could reach the hospital and have the operating room prepared. I must not be understood to say that these average mortalities set forth on a time basis, apply to every individual case. If they did they would not be averages—for we all know how the individual reaction of different patients varies. For example—a girl of sixteen, in our human series, died twenty-four hours after the onset of ileus symptoms which began forty-eight hours after removal of her appendix. Autopsy showed a pelvic abscess to which a loop of ileum was fastened by recent adhesions in a tight double angulation which absolutely occluded the lumen but gave no gross evidence of interference with the mesenteric or intestinal circulation (Fig. 1). On the other hand, an old lady of sixty-four was in operable condition at the end of seventy-two hours after onset of symptoms, survived a resection of gangrenous intestine which had been strangulated in a ventral hernia and was able to leave the hospital at the end of four weeks.

One sees the same individual variations among animals in whom obstructive lesions have been experimentally produced to simulate those of humans. For example, in a certain series of ten animals, last year (1920), one, whose intestine had been obstructed by a ligature carefully placed to avoid injury to the mesenteric blood supply, died within forty-eight hours of the onset; while another, in which an exactly similar obstruction technic had been followed, but who had several saline hypodermoclyses, was in sufficiently good condition at the end of six days to survive an intestinal resection from which he recovered. A third animal (the control in the same series) whose intestine had been transversely divided and the ends inverted, survived almost eight days without any treatment whatsoever. In another series of six animals, this year (1921), with obstructions as nearly similar as possible, one died within forty-eight hours and one lived six days with no other help than three saline hypodermoclyses. It is perfectly clear, then, that here, as in other serious sicknesses, individual variations in resistance, or—if you prefer the biological conception of disease—personal variations in the adaptability of an individual's tissues to altered environment partly determine the outcome.

Let us set down poor adaptability or—more familiarly perhaps—(1) low resistance as the first factor in the high mortality of acute ileus. This like the others is a variable factor and it includes all complications liable to accentuate it. It is for the most part beyond the surgeon's control, but a serious effort ought to be made to estimate the individual resistance in each case, and to plan or to modify the treatment in accordance with the estimate. Whether the resistance be high or low, however, there are two other factors which, if allowed to remain active, will in the end prevail over the stoutest efforts of the organism to adapt itself. They are (2) intestinal damage and (3) the formation of poisons within the intestine. Whether these go hand in hand—as one might naturally imagine—or whether they are in part independent is not as yet decided; and it is interesting to review some of the evidence one may adduce as to the independence or the preponderant effect of either.

Now intestinal damage, basically, means interference with the intestinal blood supply. It is difficult to imagine any intestinal injury that does not interfere with the blood supply. And, conversely, it is difficult to imagine any interference with the intestinal blood supply that would not result in damage to the intestine. We all are probably ready to admit that, in general, marked intestinal damage in obstruction cases is more likely to result in early death than is slight intestinal damage; that volvulus or mesenteric thrombosis is more rapidly fatal than simple, single obstruction by band or angulation. And, similarly, on observing a piece of gangrenous, strangulated intestine, we would be willing to suppose its contents to be more poisonous than those of a viable portion occluded by simple angulation. Yet we have clinical and experimental evidence to disprove the universality of such conclusions. We find exceptions to the general rule and we must try to adapt our hypotheses to explain them.

Among humans, such an exception was illustrated by the young girl whose case I cited a few moments ago. She died within twenty-four hours of onset of symptoms, yet autopsy showed an intestine only moderately dilated and with no signs of gross damage (Fig. 1). From its appearance one would have expected her intestinal contents to be relatively non-toxic; and we did indeed ascribe her early death to the complicating status lymphaticus whose lesions appeared at autopsy. Contrary to expectations, however, a moderate dose of the sterilized filtrate from her intestinal contents injected into the jugular vein of a dog proved rapidly fatal. He died within three and one-half hours with evidence of the most intense gastro-enteric disturbance shown by blood-streaked vomitus and stools, together with bloody intestinal content and a sero-sanguineous peritoneal effusion which autopsy disclosed. Here, evidently, the appearance of the intestine was no criterion for the toxicity of its contents.

A similar exception appeared in our animal series this year. Thinking to secure some highly toxic intestinal contents for a class demonstration, I made an experimental volvulus of the small intestine; and insured the

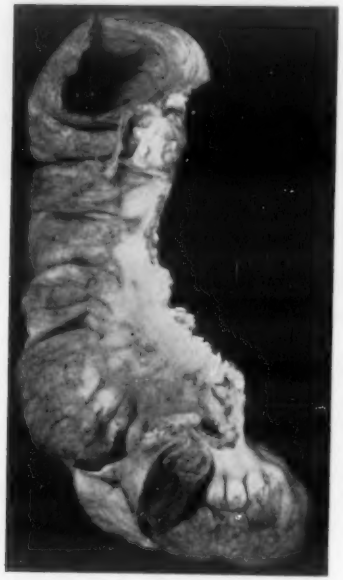

FIG. 1.—Surg. Path. No. 6085. Human ileum obstructed by adhesion and angulation. Appearance of intestine relatively normal. Contents highly toxic.

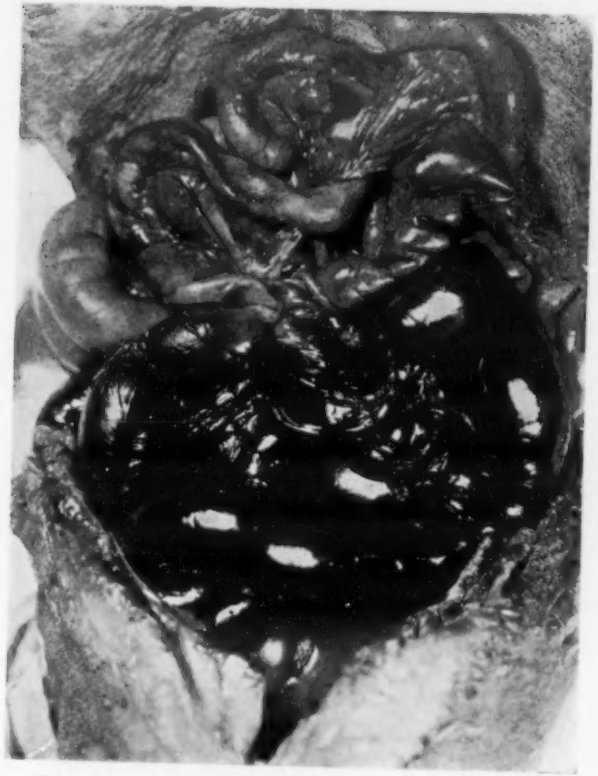

FIG. 2.—Dog No. 1698 (1921).—Experimental volvulus of ileum. Appearance of intestine gangrenous. Contents relatively non-toxic.

complete blood and contents stasis of the loop by tying a tape around its stem. The animal died within about eighteen hours. The strangulated loop was apparently gangrenous and the peritoneal cavity full of bloody exudate (Fig. 2). To our surprise and chagrin, however, the sterilized filtrate from the contents of this loop showed practically no toxicity upon injection into the jugular vein of another animal. Here, again, the toxicity of the intestinal contents and the appearance of the intestine itself bore little relation to each other.

Now, although the best supported theory at present—relating to the cause of death in acute ileus—is one which supposes the fatal absorption of poison from the intestine, it is difficult to believe that this animal died from that cause. In the first place there was no poison demonstrable in the intestinal content; and, in the second place, the drainage from the loop into the systemic circulation was absolutely shut off by the tape ligature which tightly compressed the neck of the involved loop. It is of course possible that the sero-sanguineous peritoneal exudate or transudate (whichever it is) may have been poisonous, and that such poison (if present) was introduced into the circulation via the peritoneal lymphatics with fatal result. Experiments are in train to test this hypothesis. But it is simpler to believe that—in cases of this sort—where massive gross damage is done to the intestine, its blood-vessels and nerves, another deadly factor is apparent, (4) shock.

There have been so many contradictory explanations of shock offered that we must—most of us—feel that we have still much to learn regarding its pathology and its underlying cause or causes. And it may possibly be that what I here call shock is the result of the rapid absorption, via the peritoneal lymphatics, of poisons from the intestinal wall which *may* be present *there* before they are demonstrable in the contents of the lumen. If gross intestinal damage went invariably (as it does not) parallel with the presence of demonstrable poisons in the intestinal contents, the early poison absorption theory alone would be acceptable. But I prefer to believe, at present, that an element of shock—shock from some other cause than the absorption of intestinal poison—plays a major part in the fatal outcome of certain cases of acute ileus.

This curious condition which none of us can satisfactorily explain but which all of us can sufficiently recognize under the name of shock, certainly does appear relatively early in the cases of obstruction where a portion of the intestine has had its mesenteric blood supply occluded by volvulus, mesenteric thrombosis or other mechanisms of strangulation. And, from observations upon series of cases in humans and in animals, we are fairly safe in saying that the more sudden the onset of the blood stasis and the larger the amount of intestine involved, the greater the accompanying shock.

Personally, at any rate, I am at present convinced that shock is a preponderant factor in certain cases of early death from acute ileus.

Now, in order to get an impressionistic view of the last factor which I want to consider, let us think for a moment of the human or animal body

as an oversaturated watery solution. A solution whose precipitate has so thickened and hardened at the periphery as to effectually enseal the aqueous content. Since the solution is normally of the proper density, in its different parts, to carry on satisfactorily the processes of life within the body, we may suppose that any measurable alteration from the normal density will interfere with the optimum conditions for bodily health. And, further, that if such alteration should be extended to a degree beyond that to which the body's capacity for adaptation to it extends, serious bodily damage would result.

At all events we are frequently forced to recognize the striking damage resulting to the tissues directly involved—and to the entire body—from that most rapid dehydration due to a burn. And, to a lesser degree, we can, by closer observation, recognize various instances of lesser and perhaps temporary injury to the body due to unbalanced lowering of its aqueous constituent from various causes. In acute ileus, where the output of water by urine, sweat, respiration, and especially vomitus, is frequently much greater than the intake, this type of tissue damage is often very prominent; and I want to set down, as last but not least, among the factors which predispose towards a fatal outcome in acute ileus, (5) dehydration.

Some at least of these lethal factors appear to be interdependent or to react upon each other. For example, the degree of shock appears to depend upon the amount of intestinal damage, and personal idiosyncrasy (poor individual adaptability) may determine the rate of tissue dehydration and of poison formation. But each of the factors is variable and the preponderant effect of one, in one case, and of another, in another, should not be allowed to interfere with our realization that all the factors are present in almost every case of acute ileus and that they deserve treatment.

In this paper, so largely speculative and propagandist in its form, it would be inappropriate to include the protocols of animal experiments and the analyses of case series from which arguments have been drawn or examples cited. These have appeared, or will appear, elsewhere. But it would be misleading, as well as ungrateful, to omit appreciative mention of the brilliant and painstaking experimental studies of Dragstedt, of Hartwell, of Stone and of G. H. Whipple and their collaborators whose published work has been of such value in exciting our interest in and adding to our knowledge of the ileus condition.

And, finally, having outlined the factors which, in the patient's body, must be estimated and allowed for in our plan of treatment, it would be unfair to forget those factors which we have to combat in our own minds before we can effectively translate our plan into action. No honest and intelligent practitioner can critically survey his own results or those of others in this field without feeling the conviction that he must, in future, make additional efforts to protect acute ileus patients against his faults as well as against their own failures. For timidity and delay on the doctor's part are just as fatal factors in acute ileus as any that we have ascribed to the sick man's own economy.

THE CAUSE OF DEATH IN HIGH INTESTINAL OBSTRUCTION*

By J. W. Ellis, M.D.

LIEUTENANT IN THE MEDICAL CORPS OF THE U. S. NAVY

FROM THE LABORATORY OF SURGICAL RESEARCH OF THE UNIVERSITY OF PENNSYLVANIA

Modern surgery has made rapid progress in recent years. The many aids to an accurate diagnosis, and the perfected aseptic technic, have greatly reduced the mortality of a number of acute abdominal conditions. Acute conditions such as appendicitis, gall-bladder disease, and perforation have yielded to surgical skill. High intestinal obstruction is still taking too great a toll of human life. The mortality rate in the hands of the best of surgeons is approximately fifty per cent., and if accurate statistics were available from the entire country, I venture to say that this figure would be much higher. In fact, Deaver, in a recent lecture, placed it at sixty per cent. Guillaume [71] has compiled 382 cases of acute ileus, and states that the mortality was sixty-three and two-tenths per cent.

The symptoms of high obstruction are chiefly those of a profound toxæmia, plus collapse and the symptoms of an acute abdominal storm, pain, tenderness, rigidity and vomiting. But as these symptoms point to a possible obstruction, so also do they indicate acute pancreatitis and acute fulminating peritonitis; therefore a differential diagnosis of these conditions is not only difficult but practically an impossibility.

The body is overwhelmed by the toxin and death is a matter of hours. It is true that the condition is primarily a question of mechanical interference, but surgery aimed at mechanical relief, except in the first hours of onset, is not sufficient. Richardson [51] reports that in a series of forty-six cases, operated approximately within the first forty-eight hours, he had a mortality of thirty-two and one-half per cent.; and that in fifty-four cases, operated after two days, he had a mortality of forty-eight and one-tenth per cent. He makes a plea for early diagnosis, but further observes that the great difficulty lies in the absence of physical signs confirming a suspicion of obstruction.

Surgeons differ widely as to the best treatment; some eviscerate and by means of a Monk's or Moynihan's tube empty the affected loops; some prefer an enterostomy. But whether he believes in merely relieving the obstruction, in evisceration and an emptying of the loop, or in an enterostomy, the fact remains that the results are far from satisfactory, the mortality high, and death is the result of the toxæmia. In view of these facts it becomes highly desirable to find the source and nature of the toxin involved.

There have been many theories advanced as to the source and nature of this toxin, and the method by which it produces death. Amussat,[2] in 1839, first stated that death from occlusion of the intestine was brought about by intoxication from

* Read before the Philadelphia Academy of Surgery, December 5, 1921.

the stagnating and decomposing intestinal content. Humbert,[28] in 1873, repeated this assertion. Kocher,[29] in 1877, produced a strangulation of the intestine in rabbits. At death he could not demonstrate a peritonitis; therefore he does not believe that death is caused by a bacteriæmia but by an intoxication. He also showed that the condition of the patient could be improved by removing the intestinal content and washing the stomach, or by an enterostomy. He thus gave the first experimental support to the theory of auto-intoxication.

Reichel,[53] in 1886, published the results of his researches, in which he ascribes the early severe general disturbances and death to an infection of the peritoneum. But he does not blame infection alone, because in obstruction there is an increased peristalsis of the afferent loop, while in infection there is paralysis of the intestine. At autopsy we miss the signs of a peritonitis. Kirstein,[54] in 1887, made an infusion of the loop contents, and injected 15 to 20 c.c. This produced severe gastro-enteritis, collapse, and death in from two to eight hours; when injected into the peritoneum a peritonitis resulted; intravenous injection into two rabbits caused death in from seven to twenty-four hours. He used rabbits and cats. Fifteen to thirty minutes after injection the animals showed a fall in temperature, anxious expression, hair on the back erect, diarrhœa and death. In the cats there were the additional symptoms of vomiting and convulsions; in the cats the mucosa of the colon and ileocæcal region showed ecchymoses, and these were also found scattered throughout the mucosa of the ileum. His attempts to determine the method of formation and absorption of the poison did not succeed. To determine the rôle played by bacteria he used hibernating animals—the hedgehog; in these he found the intestine sterile after six months of sleep. He then caused a strangulation and found that the second day after operation the animal was awake and took milk. The animal died on the sixth day. Autopsy did not show a peritonitis. In the portion of the intestine above the strangulation the vessels were dilated, the mucosa injected and ecchymotic; the lumen contained twenty c.c. of brownish fluid; inoculation showed bacteria throughout the entire intestine. He concluded that the results obtained were due to the initial bacteria-free condition of the bowel, which later became infected from the milk. The toxic action of the content of the afferent and efferent loops did not differ, though the conditions for the development of the toxin were better in the afferent loop.

Bouchard,[4] in 1887, assumed that death was caused by an auto-intoxication and upon this hypothesis he would explain the general symptoms that arise in ileus and in incarcerated hernia. He compares the similarity of the symptoms in these conditions to those of cholera, and for further proof calls attention to the presence of an albuminuria. Bokai,[7] in 1888, took as a basis for his experiments the fact that fatty acids in the intestine act as irritants, causing peristalsis, and that if, for any reason, they are produced in excess, as in obstruction, they cause increased peristalsis and symptoms of inflammation of the wall of the gastro-intestinal tract. He states that the higher degree of intoxication is brought about by the products of putrefaction of the protein.

Talma,[62] in 1890, sought the cause of death not in inflammation nor in anatomical lesions nor in a disturbed function of the diaphragm. From his experiments he concludes that the intoxication in these and similar cases is caused by the disturbance of the entire organism, and that the immediate and distant effect of the overfilling of the stomach is the reason for death in ileus. Kraft,[31] in 1891, found that after strangulation cultures of the heart-blood and peritoneum remained sterile, and concluded that death is due to an intoxication. Reichel,[53] in 1892, rejected his former views and maintained that in straight occlusion, in spite of the distention present, there was no passage of bacteria through the intestinal wall. Nicholaysen,[42] in 1895, collected the intestinal content from a case of obstruction, filtered, and

injected into a mouse and a guinea-pig; it killed in each instance. He cultured the heart-blood and peritoneum and found both sterile.

Gley and LeBas,[24] in 1897, used Witte's peptone and decided that the immunity resulting from injections depended upon the dose per kilo; they failed in their attempts to confirm the statement that a preliminary injection confers an immunity for twenty-four hours. They state that proteose injection caused an increase in lymph flow, and decided that the toxin is not a proteose because it did not possess a lymphagogic action. Chittenden, Mendel and Henderson,[8] in 1898, reported that after the injection of a proteose the coagulation time of the blood is reduced and that there is a temporary immunity produced but this immunity is not complete, lasting only about an hour. Chittenden[10] further reports that peptones and proteoses cause a fall in blood-pressure and a distintegration of the leucocytes. This disintegration releases nucleo-protein and histon, with the production of a clot-retarding substance. Proteose injection caused an acceleration of the lymph flow; if the lymph was collected, then the clotting of the blood was barely affected, while that of the lymph was delayed, and the lymph changed in character, the lymph becoming reddish in color.

Nesbitt,[18] in 1899, made a study of the intestinal content above the occlusion of the small intestine of a dog. He found that if the animal was fed food rich in lecithin he obtained cholin and neurin, and believed that the neurin was the lethal factor. Buchbinder,[1] in 1900, showed that the obstructed loop was rapidly filled, not only by the content but by a more or less severe transudation into the lumen, especially if the venous flow were interfered with. If both veins and arteries were compressed, transudation did not occur. It appears that the intestinal wall is not permeable to bacteria or microörganisms, and he is convinced that he has shown in a conclusive fashion that even disturbances of the circulation to a high degree do not permit the passage of bacteria through to the serosa and into the peritoneal cavity. For this, wandering through deeper injuries are necessary, and the beginning of irreparable injury with consequent gangrene first permits this to occur. He repeats that the intestinal wall first becomes permeable after injury and gangrene.

Kukula,[29, 30] in 1901, in his first experiments occluded the intestines of dogs, and injected the contents into cats; he also obtained the material from ileus patients and injected this into animals. He found that these contents were toxic, and not specific for species. He first assumed that the symptoms were reflex symptoms, due to irritation of the nerves of the intestine. The second assumption was that auto-intoxication was the causative factor of the symptoms, but he states that this is only an unsupported hypothesis. Third, he investigated the normal intestinal content and the urine. He divided the toxic substances found into two groups, arising in normal or excessively increased processes of breakdown, according to whether it is due to a breakdown of the carbohydrates or to the putrefaction of the proteins. The nature of the toxic substance is obscure, but it may be advantageously extracted with amyl alcohol. This extract is as toxic as is the first filtrate of the intestinal content, so the toxin is unchanged by the method of extraction. Borszeky and Generisch,[6] in 1902, published a report of their experiments, in which they attributed the toxin to the bacillus coli, and state that this organism is present in the blood and peritoneum before there is any damage to the intestinal wall. Albeck,[1] in 1902, tested the wall of the loop as to its permeability to bacteria; he concluded that death is due to a toxin. He found at autopsy that the intestinal mucosa is ecchymotic. The work with the toxin showed that it is soluble in water, resists boiling, and passes through a Chamberland filter. He concludes first, that death in strangulation not infrequently occurs without peritonitis, and results exclusively by poisoning from the intestine; second, that the site of the formation of the toxin is not only the afferent intes-

tine, but also the strangulated loop, and in many cases is found chiefly in the loop.

Wrzosck,[46] in 1904, concluded that bacteria arising from the intestinal tract get into the mesenteric glands and other organs. He found that if he fed prodigiosus cultures and then tied the thoracic duct, he could not recover the organism in the blood or organs. Clairmont and Ranzi,[4] writing in the same year, stated that the kind of animal used for the injection of the toxin exercised no real influence upon the result, provided only the proper proportion between the body weight and the dose of poison used were maintained. They filtered the intestinal content through a Pukal or Reichel filter, and tested the filtrate for bacteria; if these were present, the material was again filtered. They found the same toxin in the content of human ileus cases as in the content obtained from the obstructed loop of animals. They studied the rate of resorption of the upper intestine, and found that it is more active up to the first half of the time the animal lived; after this it is slowed. They made bouillon cultures of the content in ileus under aërobic conditions, and obtained the same result as from the original content. Cultures made under anaërobic conditions failed to show the toxin. They state that the toxin possesses hæmolytic, cytolytic and heat-resisting properties. In their opinion the toxin is bacterial in origin, different poisons arising from different organisms, and there may be a symbiotic relationship.

Charrin[13] at this time published his conclusions, in which he maintains that in its normal state the digestive juice contains innumerable poisons. Helmburger and Martini,[28] working with transudates, state that in the solution of this problem, one is confronted by difficulties, chiefly those of reproducing the disease, and especially by bacterial contamination of the material. They show that slight disturbances produced in the intestinal wall by interference with the circulation are not sufficient to render the intestinal wall of rabbits permeable to bacteria, and that the musculature, apparently, offers the greatest resistance. They conclude that absolute necrosis is necessary, and that lesions that have lost their impermeability to bacteria cannot be recovered from.

Magnus-Alsleben,[41] in the report of his results of his investigations, states that in the content of the upper small intestine of a dog, as well as in the mucosa, there is found, after feeding various kinds of meat, and apparently after feeding bread, fats and starches, but not after feeding milk protein, a toxin substance; this toxin produces in rabbits, upon intravenous injection of the smallest doses, a general central paralysis with subsequent convulsions; death generally resulted from a stoppage of respiration. Many times during the period of paralysis, rapid recovery took place, after which the animal was immune for several hours to further injections. This effect does not follow the injection of similar doses into the portal system. Boiling in an acid solution destroys this substance. He states that the content of the entire small intestine contains, after every kind of diet, a substance which in the smallest doses produces an immediate and rapid fall in blood-pressure; this effect lasts for one minute at the most. This depressor substance is not detoxified in the liver, but is destroyed by boiling in an acid solution. He rules out the pancreas as the source of the toxin, having tested the juice obtained from a pancreatic fistula; but calls attention to the fact that the fluid flowing from such a fistula contains only trypsinogen, and no active trypsin. He states that the toxic effects are like neither neurin, proteose or peptone, nor the basic products of protein splitting alone. He believes that the intact mucosa and the liver act as retoxifying agents.

Roger and Garnier,[47, 48, 49, 50] in 1905 and 1906, first used rabbits and determined the coefficient of toxicity. They found that ligation of the rectum was not as toxic as a higher ligation. In perforation the toxicity became sixteen times normal. They caused peritonitis by injecting anaërobic organisms, and obtained a toxicity twice the normal. They made a solution of the intestinal content in

alcohol, and found that the material dissolved was not toxic, but that a watery solution of the precipitate produced diarrhœa. This watery solution, however, was less active than the primitive liquid. They next used dogs, and after a fast of forty-eight hours, fed them 100 to 1000 grams of meat. The dogs were killed and the contents measured. This content was passed through linen, centrifuged and filtered. The extremes of the lethal dose were from 0.41 to 3.4 c.c. per kilo. They found the blood liquid and increased the resistance by injections of an extract of leech. The duodenal content was most active; injection into a peripheral vein was 0.14 times more toxic than injection into the portal vein. They found that the material dissolved in alcohol had a high toxicity for dogs, and decided that there must be a plurality of toxins. The alcoholic solution caused death without convulsions and increased the lymph flow; the watery solution of the alcoholic precipitate caused diarrhœa and death. They found that the toxicity diminishes if the animals are fed milk, and is one-ninth as toxic as after meat feeding. They next made extracts of the different portions of the gastro-intestinal tract; they found that the stomach and cæcum had an equal toxicity, and were less active than the duodenum, jejunum and ileum. The colon was more toxic than the stomach and cæcum, and had the property of producing hemorrhages in the Peyer's patches; this did not follow injections of any of the other preparations. Extracts of the ileum and appendix caused clotting in the right heart; an extract of Peyer's patches showed this clotting power, but it was lost in the remainder of the ileum. They state that the toxicity has no relation to putrefaction, and cite, for example, that the duodenum is more toxic than the large intestine. They injected the pancreatic secretion, and found it non-toxic. The duodenal content was slightly toxic, when mixed with pancreatic secretion, the mixture became quite toxic. They believe that the toxicity of the intestinal content depends upon the food, and that the secretions are only slightly toxic. They found that after feeding meat, if there was a large amount of the gastric juice present in the duodenal content, this content was less toxic; they found that incubation increased the toxicity, and they have, by these different processes of treating the content, been able to modify profoundly its toxicity. They conclude from their experiments that as a means of defense the liver is only slightly active; that the most important defensive rôle is played by the intestinal mucosa; this must be intact from the point of function, for if it is impaired, or its vitality modified, autolysis results.

Von Khautz,[48] in 1908, concluded that the supposition of pure bacteriæmia as the cause of death in mechanical and paralytic ileus, without a simultaneous peritoneal infection, is improbable. It is to be assumed in such cases that death is caused by toxic substances in the intestinal content.

Draper-Maury,[49] in 1907, regarded it as conclusively demonstrated that death following duodeno-jejunal obstruction, and probably acute pancreatic disturbances other than infections and traumatisms, and death associated with symptoms known as acute gastric dilatation, and following certain operations on bacterially clean surgical fields in which the hepatic ducts have been invaded, all these now uncorrelated and little understood deaths due to a common, underlying cause as yet not defined. He believes that death in ileus is a physiologic one. Physiologic death comprises not alone that which follows certain substances by removal of their secreting cells, but also that brought about by mechanical interference with detoxication of the normal secretions of the body. This would suggest an internal secretion of the duodenum. He starts with the hypothesis that obstruction of the lumen works no ill to the organism save through an interference with the physiologic exchange or balance of the duodeno-jejunal secretion.

Cybulski and Tarchanoff,[51] in 1907, stated that the toxicity depends upon the secretions poured into the upper intestine, especially the pancreatic juice.

Braun and Boruttau,[3] in 1908, stated that they did not believe that a poison existed, because tracings taken just before death did not show convulsions, arrhythmia or other stormy manifestations pointing toward a poison. On the contrary, everything pointed toward a gradual extinguishing of life. The increase of resorption in the afferent loop of intestine, assumed by Clairmont and Ranzi[8] in the first nine hours after the production of an occlusion, they do not consider correct; on the contrary, the resorption is generally slowed from the very beginning, as they have determined by the injection of strychnia into the lumen of the intestine above the obstruction. Indeed, the delay in many cases was so great, the resorption so slow, that an otherwise absolutely fatal dose is not able to bring about death. Where no infection is demonstrable and death nevertheless occurs, they hold that the severe functional disturbances in the abdominal cavity suffice to cause death by causing a cerebral anæmia, due to bleeding into the splanchnic area. They further state, in their discussion of the toxicity of the intestinal content, that the truth cannot lie in the assumption of the effect of bacteria and putrefaction; but one must sooner think of the breakdown products of the food by the intestinal ferments, or indeed, of these ferments themselves.

Draper-Maury,[25] in 1909, stated that the bile is in no way connected with the cause of death, but considers the pancreatic secretion the lethal agent. Sauerbruch and Heyde,[61] in 1909, produced parabiosis by uniting the peritoneal cavities of two animals, and then produced an ileus in the one. They ruled out the possibility of peritoneal infection going from one to the other. A few hours after operation the temperature in both animals rose; later the temperature of the operated animal fell, but its parabiotic mate preserved an increased temperature up to the death of the operated animal; if the living animal was then removed and sewed up, the temperature returned in a few days to normal. This is explained by the relative amounts of toxin, in that the second animal only gets sufficient amounts of toxin to cause the rise of temperature, and not the fall and the fatal effect.

Combe,[12] in 1909, was an ardent adherent of the auto-intoxication theory. He assumed a threefold protection against intoxication, *i.e.*, the intestinal mucosa, the liver, and the antitoxic glands, the hypophysis, the thyroid and the adrenals. Dale,[16] in 1910, compared the action of Witte's peptone, the effect of which had been considered by some to be analogous to the effect of the obstruction toxin, to that of his Beta-I, or histamine, and considered that the effects of the peptone were similar to, but not necessarily identical with, the effects of the histamine. Esau,[21, 22] in 1910, found that if one resected a loop of the intestine, leaving its mesentery intact, closed the ends and transplanted this loop beneath the skin, having restored the continuity of the intestine by an end-to-end anastomosis, symptoms appeared, and death might result. If one then loosened the suture of one end of the excluded loop, large amounts of foul content escaped and the dog recovered rapidly. This, he holds, strongly proves the autointoxication theory. He states that resorption diminishes with the course of ileus, but that a short period is sufficient to overload the animal with toxin arising from the stagnating content. He thinks that relatively small amounts of toxin would suffice to rapidly produce an irreparable injury to an organism on which other different harmful factors are working. He concludes that it is not possible to have closed, excluded loops in the abdominal cavity or extra-peritoneally. Wilms,[65] in 1910, concluded that the symptoms were due to reflex nerve irritation.

Murphy and Vincent,[39] stated that interference with the circulation of the obstructed intestine is the vital factor in the production of the symptoms of ileus. Enderlein and Hotz,[20] in 1911, presented their resorption theory in peritonitis and ileus. Their results show that the resorption power of the obstructed loop is considerably lessened and point to the conclusion that the portion of the intestine concerned in ileus is harmed in its resorptive power. Using five per cent. sodium

chloride solution, they found that there was an increased excretion into the loop in ileus; the quantity may reach double that found in the efferent loop. Their work shows another important result, in that they found that the power of resorption of the entire intestinal tract is lessened.

In 1912, 1913 and 1914, Stone, Whipple and Bernheim,[54, 55, 56, 57] published a great many papers on the results of their investigations. In their earlier work they found that death occurred usually within forty-eight hours; a microscopic study of the organs of these animals showed them to be normal. They showed that the occluded loop is not essential to life, and that drainage of an occluded loop plus irrigation, may save life. They adopted the secretion theory as the most probable, and scout the circulatory disturbance theory of Murphy and Vincent. They found that the toxin is not destroyed by heat, autolysis, or pancreatic digestion; it resists putrefaction for a few days, and it does not stimulate pancreatic secretion, as does secretin. Blood from animals moribund from the effects of an obstructed loop is not toxic to normal animals. Autolysis of the normal mucosa did not produce the toxin. They believe that sub-lethal doses protect against large doses, and probably against the closed loop. In 1913 they showed that the toxic substance is absorbed from the mucosa, and not from the lumen. The normal mucosa did not contain this toxin; destruction of the mucosa prevented the formation of the toxin. In one experiment noted (56, p. 315), the entire intestinal mucosa was removed from a dog fatally poisoned, autolyzed for seven days with toluol; upon injection, it proved inert. In 1914 they showed that with circulatory disturbances, food derivatives, gastric, pancreatic and biliary secretions excluded as a possible source, the dogs died with the characteristic symptoms; the autopsy findings were typical, and suggested an intoxication. The content of the closed loop is highly toxic when injected into normal dogs, and the reaction resembles that of dogs with a closed loop. They hold that the toxin is formed by the mucosa of the closed loop, and that resorption from the mucosa is the prime factor in the intoxication. The cause of the perversion of the secretion is not explained. They believe it possible, by using sub-lethal doses, to confer an immunity, and that this immunity is produced by the action of the parenchymatous organs. They have demonstrated that absorption of the toxin from the lumen is a negligible factor.

Hartwell and Hoguet,[26, 27] in 1912, maintained that the symptoms were caused by dehydration of the tissues, with resulting disintegration, and that if the water were replaced, life would be prolonged; but that if the distention of the bowel above the obstruction became so great that the mucosa was damaged, the dogs could not be kept alive with the salt solution. McLean and Andries[30] believe that death in high obstruction is not due to a toxæmia, neither from bacteria nor altered physiologic secretion. They state that a depletion of the vascular and lymph system, causing a grave disturbance in the circulation, is a prime factor, and that a pathologic change in the sympathetic nervous system, probably a loss of control, is a contributory cause. McKenna,[35] in 1913, held that the toxin is the result of a disturbance of the physiologic balance of the normal intra-enteric secretion from the duodenal mucosa. The fatal factor in general peritonitis may be due to this duodenal secretion; the results are the same whether the ileus is produced by mechanical obstruction or by paralysis, and he recommends an early jejunostomy.

Sweet,[48] in 1913, called attention to the relationship existing between the pancreas and the adrenals, and to the striking clinical resemblance between high obstruction and acute pancreatitis. Draper,[14] in 1914, repeated his assertion that the cause of death was not bacterial, but truly autotoxic, from the cells of the epithelium. A microscopic study of the heart, the liver, the kidneys and the intestine showed only a capillary dilatation. He showed that the decrease of the

water content of the tissue of dogs with a duodenal obstruction is about equal to that following pilocarpin. These toxins are partly eliminated by the stomach and the colon. He found that if the animals with obstruction were fed the epithelial cells of the small intestine of healthy animals, they survived nearly twice as long. Davis,[17] in the same year, working with cats and dogs, found that the cats exhibited a higher immunity to the closed loop toxin; also that the dogs would react to the toxin obtained from the cat.

Murphy and Brooks,[27] in 1915, in summing up their work, report that they believe the toxin to be produced by bacterial growth, and that it may be formed in any portion of the intestinal canal or in the gall-bladder. Its mode of entry into the circulation is by way of the thoracic duct, and an interference with the circulation is an essential factor in allowing absorption. Sweet,[28] in 1916, stated that the finding of the poison in the isolated loop is no proof that it is formed there, as it is possible that it is formed elsewhere and then excreted into the loop. He, with Peet and Hendrix,[29] question the statement of Whipple and his co-workers as to the formation of the toxin in the obstructed loop, and state that it may be formed in the afferent loop, which was functionally obstructed in the experiments reported by Whipple and his associates, and then be excreted into the obstructed loop. The destruction of the mucosa of the isolated loop with sodium fluoride proves nothing, because by destroying the mucosa, one destroys the excreting agent.

Whipple, Rodenbaugh and Kilgore,[30] in 1916, attacked the dehydration theory of Hartwell and Hoguet, and cited as proof against this theory that dogs which had been dehydrated with pilocarpine and purgatives showed no signs of intoxication. In their studies of the properties of the toxin, they found that it did not produce anaphylaxis in the guinea-pig; that it produced a slight immunity; that it was more or less removed by a Berkefeld filter; that autolysis with normal intestinal mucosa destroyed it only after a period of eight to twelve months; that it resists pancreatic and ereptic digestion, and in these respects it resembles a hetero-proteose.

Dragstedt, Moorhead and Burcky,[31] in 1917, stated that they did not believe that the toxæmia was the result of increased absorption; and for evidence against the bacterial origin and the theory of cellular activity, they point to the fact that it is not manifest in typhoid, dysentery and ulcer, nor is it manifest in congenital atresia until after feeding takes place. They experimented with sterile closed loops, using as an antiseptic agent sterile water and ether. Of twenty-five dogs thus operated upon, sixteen died of perforation and peritonitis. A few of the remaining were killed, and the examination of the fluid content of the loop showed bacillus coli. One case of perforation did not show a peritonitis. In another series they prepared the sterile loop, and occluded the blood supply. In one case autopsy showed that the loop had disappeared; in the second case death resulted from necrosis of the loop. When the loop was of the duodenum, death resulted from the sterile loop in apparently the same time as from the non-sterile loop. They give as possible factors in the production of death in the sterile duodenal loops, first, as the duodenum is chiefly secretory, they were unable to establish an equilibrium between secretion and absorption, and second, the arrangement of the blood-supply differs, so that a slight distention causes occlusion. They assert that these factors, and not any peculiarity of the secretions of the glands or of bacterial flora, is the cause of death. They ruled out, to their satisfaction, the question of the toxicity of the normal secretion, perverted secretion or aberrant activity of the cells, by isolating a loop, sterilizing it, and dropping it into the abdominal cavity, leaving the ends open; fifty per cent. of the animals lived. They conclude that bacterial activity, plus necrotic tissue, or the absorption of the toxic products resulting from the action of putrefactive bacteria upon necrotic

tissue, is the important factor in the rapid death in simple, closed intestinal loops.

Davis and Stone,[18] in 1917, publish the result of work showing that the toxins that produce the same symptoms and the same lesions of the intestine as do the toxins derived from an obstructed loop, may be elaborated from the intestinal secretion in vitro. They collected first the washings of an isolated loop over a water bath, at a temperature of 90° to 95° C. This was then heated to this temperature for one-half hour on the two succeeding days; when injected it did not produce symptoms or a change in blood-pressure. The unheated secretion was non-toxic, as was that kept under toluene and chloroform. The unheated loop washings were collected in a sterile flask, and kept as well as possible from contamination at 37° C. for eighteen hours; these showed a profuse bacterial growth, and upon injection after filtration through a Berkefeld filter, this caused death with a typical picture in six hours. They do not say positively that the bacteria are responsible, but they do claim to have ruled out enzyme action and the perverted cell theory.

Whipple,[19] in 1919, in summing up the results of the work done by him and his associates, states that nothing produced within the intestinal tract can be directly concerned in the intoxication of intestinal obstruction. The intestinal epithelium is impervious to all toxic substances which can be demonstrated in any amount in the material accumulating in the obstructed intestine. Material obtained from the unobstructed intestine can be fed in unlimited amount, injected into the duodenum, or into the closed loop, without causing the slightest degree of intoxication. He believes that the toxic substance or substances can be formed only by the epithelium of the mucous membrane of the small intestine under obstruction conditions. This material, absorbed by the blood, causes the characteristic symptoms. The poison is not found in the normal intestine. Closed colon loops are never associated with any definite clinical intoxication referable to the closed loop.

Cannon, Dragstedt and Dragstedt,[7a] in 1920, endeavored to support the conclusion of Dragstedt, Moorhead and Burcky [19] that bacterial activity plus necrotic tissue is the important factor in the production of the toxin. Their experiments, directed toward the changing of the bacterial flora of the intestine, do not seem, however, to have led to any clear-cut result.

In a critical examination of the work of the above authors, one thing must be constantly borne in mind; they have manifestly not all been dealing with the same type of poison. One group is evidently dealing with a poison which violently attacks the central nervous system, while the other group deals with the toxin which the more recent writers have discussed to the exclusion of the nerve poison. The toxin under discussion does not produce convulsions, etc., but produces symptoms which point to a primary attack upon the gastro-intestinal tract, as evidenced by the vomiting, retching, diarrhœa and tenesmus, with a secondary pronounced effect upon the blood-pressure.

After reviewing the literature upon the origin and method of attack of this toxin, one cannot but be impressed by the number of theories advanced, and the recurrence of these theories in cycles, as it were. The earliest,[2] and also the latest,[19, 7a] was that of auto-intoxication arising from the stagnating and putrefying intestinal content. While this was at first only an hypothesis, it early received some experimental support. This experimental work, however, did not go far enough, as the authors were content

to reproduce the condition of ileus, and after the death of the animal, the presence of a dilated proximal loop containing a material which, when injected, caused death, was considered to have solved the problem. This theory has been disproved by many workers and in many ways, the most conclusive proof being that which we will offer of the speed with which the toxin appears in the intestinal content of a perfectly normal animal, after the intravenous injection of the toxin.

The next theory to engage the attention of many workers for a time was that dealing with the action of bacteria under the condition of obstruction; these held that death was the result of a bacteriæmia. This theory was disproved by the finding, in so many cases of ileus, that the heart-blood, peritoneum and organs were sterile.

The third theory deals with the secretions poured into the upper intestine. Better technic has enabled later observers to rule out the pancreas and the biliary apparatus as the source of the toxin, even though we shall show from our experiments that the toxin appears in the intestine of dogs dying from an acute pancreatitis.

The fourth theory ascribed the death to the cerebral anæmia which resulted from the bleeding into the splanchnic area, or, in other words, "shock." This is disproved by the many clinical conditions in which "shock" is observed, yet the symptoms and the physical findings do not resemble ileus, and yet we shall show that the toxin appears in the intestine following the splanchnic congestion which results from portal thrombosis.

The fifth theory, that of death being due to reflex irritation of the sympathetics, is not grounded upon facts produced by experimental work.

The sixth theory advanced is that a disturbed circulation is responsible for changes in the intestinal wall which produce the attending symptoms and death. This is disproved by the fact that intravenous injection causes death with typical symptoms in a normal dog—*i.e.*, without at least any gross mechanical changes in the circulation.

As before stated, all these theories have had or still have their adherents. They individually seem to have a semblance of proof which is often difficult to disprove, because of the extreme complication of the conditions under which one must work. But a careful analysis shows that no one of these theories offers a sufficient explanation of the symptoms and findings.

The seventh theory is that of a perversion of the normal function of the cells of the duodenal mucosa. This idea was first expressed by Braun and Boruttau,[3] Draper,[14, 15, 40] and Whipple, Stone and Bernheim,[54, 55, 56, 57]. Though this theory has been questioned, it has yet to be actually disproved.

The eighth theory is that dealing with the rapid and extreme dehydration of the tissues. This was first advanced by Hartwell and Hoguet,[26, 27] and has lately been restated by Bacon, Anslow and Eppler,[72] and by Stone.[73] These last have ingeniously tried to use the fact that in some instances there is a rise in the non-protein blood nitrogen, due, as they state, to the water loss, to support the theory of dehydration death. This theory seems hardly

tenable in view of the fact that death may follow the intravenous injection of the poison before any loss of water has occurred.

This brings us to our own entry into a study of the problem. We were impressed by the extremely close similarity existing between the clinical pictures of acute pancreatitis, ileus and acute fulminating peritonitis. We became convinced that such a similarity could arise from the fact that these clinical entities had a closely related factor as the cause. We do not wish to be misunderstood and have one think that we believe that one single factor may cause either ileus or acute pancreatitis, but we do believe that the toxin that is the cause of death in the one case is identical with, or closely allied chemically to, that which produces death in the others, irrespective of its initial source. We believe that it arises within the cells of the mucosa of the duodenum in ileus, and takes origin in the cells of the pancreas in acute pancreatitis. It is inert in the cells in the normal organ, and when it is excreted into the lumen, under normal conditions, is immediately combined with the content of the intestine, and is innocuous.

In obstruction the condition is changed, and instead of being thrown out into the lumen of the intestine, the major portion is forced into the lymph, and thence into the general circulation; the portion thus excreted is intensely toxic, judging from the toxicity of the relatively small amount that is present in the content of the bowel.

The method of obtaining, and to a certain extent, of purifying the toxin, is a modification employed by Wells and Osborne [67] in their work with vegetable proteoses. The content and mucosa of the small intestine was collected in approximately 250 c.c. of hot water. This was thoroughly mixed, and strained through gauze and cotton; to the solution was added five times its volume of ninety-five per cent. alcohol. The precipitate was filtered off, and boiled with 100 c.c. of distilled water; after all traces of alcohol had disappeared, one gram of magnesium sulphate was added, and it was allowed to boil for a few minutes. The mixture was filtered, and the filtrate precipitated in five times its volume of ninety-five per cent. alcohol; this was filtered, and the precipitate dried in a desiccator. By this method, we had an easily handled, stable product, and were satisfied that all bacterial action, once the original material had been removed from the intestine, had been eliminated. Before using, the precipitate was dissolved in from twenty-five to fifty c.c. of distilled water, and dialyzed for two hours against distilled water.

At first we used material obtained from a series of closed loops. When engaged upon another problem in which complete adrenalectomy had been performed, we noticed that at autopsy the small intestine presented a picture grossly identical with that of the obstructed intestine. This material, prepared in the routine way, produced exactly the same symptoms as the other, and at death the autopsy findings were the same. We also prepared the material found in the intestine of dogs upon which an Eck fistula had been performed, but because of too small an opening between the veins, clotting

had occurred, and death had resulted from a portal congestion. This product also reproduced the symptoms and the gross pathology of obstruction.

As we have mentioned above, our first method of obtaining the toxin was to make an isolated loop and restore the continuity of the intestine by an end-to-end anastomosis. At death we found that the loop had ruptured, or was greatly distended, and showed an acute congestion. The contents, with the mucosa, were prepared by the technic described, and were injected intravenously. The symptoms of poisoning were violent vomiting, retching, diarrhœa with tenesmus, progressive prostration, and frequently death. We obtained the material from six of these high loop animals, and injected normal animals; of these injected dogs, three died with characteristic symptoms, and the autopsy presented the typical picture of high obstruction. From the intestinal content of these non-operated dogs, killed by intravenous injection of the poison, the toxin was prepared and injected into other normal dogs, with the same result.

To rule out the possibility of a histamine reaction, guinea-pigs were injected with this toxin. As the guinea-pigs did not show any reaction whatever, we considered that histamine played no part in the production of the symptoms and death.

Fourteen adrenalectomies were performed. In a few of these cases the duct of Santorini was ligated, and a cannula was tied into the duct of Wirsung, which drained into a rubber bag, in an effort to obtain the pancreatic secretion separate from that of the intestine. Toxin was obtained from both series alike, regardless of the drainage of the pancreas. Upon injecting this toxin the following striking results were noted; the dogs died in a short space of time with the usual symptoms characteristic of the toxin of high obstruction. One dog varied from the usual course in that it lived for three days following the injection; the autopsy in this case showed areas of liver necrosis, the stomach contained several patches of hemorrhage, the intestines were intensely congested and hemorrhagic throughout the entire length. A third dog, in which death had occurred within the usual time, presented a marked pathology; there was free blood in the peritoneal cavity, apparently from a rupture of what looked to be varices beneath the capsule of the liver; the gall-bladder was intensely œdematous, the walls being one-fourth of an inch thick; the pancreas was œdematous in that portion lying along the intestinal wall; the liver lobules were clearly marked; there was blood in the lower small intestine, and congestion of the upper end of the duodenum; the valvulæ conniventes of the large bowel showed hemorrhages. In one dog after the adrenalectomy with pancreatic fistula, death followed the usual symptom-complex; the autopsy, however, presented a different picture; the bag had slipped off the cannula, allowing the pancreatic secretion to escape into the peritoneum, and we found as a result a general peritonitis with free bloody fluid, fat necrosis in the omentum and along the gut in the neighborhood of the pancreas; there were areas of necrosis in the liver; in the antrum of the stomach there was a clean-cut ulcer; there was an acute pancreatitis; the intestinal mucosa was congested and hemorrhagic. Material obtained from the intestine of this dog gave a highly toxic preparation.

We next took the content of the intestine of a dog that had died from the intestinal form of distemper. The material obtained caused, upon injection, the immediate death of the animal. The content of this intestine was removed and

prepared as soon as death had occurred; injection of this material caused death with the symptoms and gross pathology resembling high obstruction. The material from this animal upon injection caused such a violent reaction that we decided to kill the animal. The autopsy showed considerable areas of necrosis of the gastric mucosa, necrosis of the liver, and a typical intestinal picture. The preparation from this dog, though it caused a marked reaction, did not kill. We have in this series carried the toxin through four dogs, causing the death of three with typical symptoms and characteristic autopsy findings; in the fourth animal, though the symptoms were present, the dog recovered.

We next decided to see whether the intestinal contents of dogs dead from acute, non-bacterial peritonitis, contained a toxin. Under strict aseptic conditions, the pancreas was removed from one dog and dropped into the peritoneal cavity of a second dog. This is known to produce a condition of acute pancreatitis with acute, fulminating peritonitis, but in which the bacterial element, which has been such a disturbing factor in the entire history of the study of this problem, could be ruled out. Autopsy following the death of the animal showed an acute peritonitis of an extreme grade; the intestinal content was prepared and injected into a normal dog. This caused the usual symptoms and death, and autopsy showed the usual pathological picture.

The next part of the problem was to determine the state of the normal content. The content was taken immediately following the death of five dogs, killed by ether or by hemorrhage, and prepared in the routine manner. These preparations, when injected into normal dogs, gave only a very doubtful reaction, *i.e.*, defecation.

The next factor to be considered was the rôle played by autolysis; the intestine of a dog killed by ether was allowed to stand at room temperature for seven hours; at the end of this time the content and the mucosa was removed and treated in the routine way. Injection caused a very mild reaction. The intestines of four dogs were allowed to stand at room temperature, in the one case four hours, in the other three cases five hours. A preparation of the content in the first case did not produce the reaction, in the other three cases it caused death. In these cases the bacterial content of the intestine was not interfered with. As a control the intestines of three dogs killed by ether were washed out with water and allowed to stand at room temperature for five hours; the content was then prepared, and injected into a normal dog. This did not produce a reaction. Next the intestines of four dogs killed by ether, were washed out and filled with chloroform, and allowed to stand at room temperature for five hours; the content and mucosa was then removed, the chloroform driven off, and the material prepared in the routine manner. Upon injection of the entire product into one dog, no reaction occurred. We then took the intestines of four dogs killed by ether, and after washing out the content, filled them with water and allowed them to remain at room temperature for five hours; the content in these intestines was noticeably greater than in the cases where chloroform was used. The content of these four together, prepared in the usual manner, and injected ino a normal dog, did not produce any reaction. These results, when taken with the results reported by Davis and Stone,[18] raise the question of whether the normal intestinal content, in the presence of the normal bacterial content of the intestine, may possibly give rise to the toxin on standing. This problem reminds one of work which has been done on the activation of the pancreatic juice by bacteria; if, as some have maintained, pancreatic juice can be activated by bacteria, then it would be necessary to admit that the proteolytic ferment of the intestine itself could likewise be activated by bacteria. Nevertheless, in view of the above experiments in which the toxin appears so rapidly in the lumen of the intestine after intravenous injection, we do not believe that bacteria have an exclusive rôle in the production; indeed, we believe that we must conclude that they are not even necessary to its production.

We next determined to try the effect upon the toxin of erepsin, a ferment found chiefly in the lower portion of the small intestine. The erepsin used was obtained from Parke, Davis and Co. Three grams of the erepsin were extracted with fifty c.c. of water for two and one-half hours of body temperature. The mixture was then centrifuged, and the supernatant fluid used for injection. Four such preparations were made and injected into four dogs; only one of these animals showed a slight reaction. Four toxin preparations were combined, and one-quarter of this product was injected into each of two dogs; both died. The remainder was divided into two equal portions; to each was added 0.25 gram of erepsin, and they were incubated for two and one-half hours at body temperature. Two dogs were injected; in the one case the symptoms preceding death were more violent than in the two control dogs; in the other there was no apparent change in the picture. At autopsy the first dog showed a perforated duodenal ulcer, free blood in the peritoneal cavity, marked œdema of the gall-bladder and pancreas, while the autopsy in the second dog did not deviate from the usual picture. Of nine other dogs, injected with this toxin-erepsin preparation, made as above, there were three deaths, five gave a marked reaction, and one did not respond. To summarize this portion of our work, we find that the toxin-erepsin combination caused five deaths, five were profoundly affected, and one was only slightly affected. These results discredit the theory that the lower intestine exerts a detoxifying action. They also tend to show that the toxin is neither a proteose nor a hetero-proteose, since both are digested by the tryptic and ereptic ferments.[82a, 70]

Our next efforts were directed toward the action of repeated sublethal injections, increasing in amount, to determine whether they would produce an immunity, or whether they would cause a picture of chronic intoxication with ulcer formation. A dog was given 0.75 g. of toxin, to which he reacted violently; after one week the dog received 1.0 g. of the toxin; this did not produce a reaction. A week later, two grams of the toxin were injected; there was no immediate reaction, but the dog died the following day. The autopsy showed the following picture; the duodenum and upper jejunum showed slight indication of the usual involvement; the areas of submucous hemorrhage increased to the terminal ileum, which showed a considerable involvement; the ileocæcal valve was hemorrhagic throughout its entire area; the cæcum was hemorrhagic, and had one area of ulceration; the large bowel contained old and new areas of submucous hemorrhages. A second animal which had received a sublethal dose was promptly killed by a second injection; the autopsy showed the usual picture. This shows that the production of an immunity is very dubious, as one would expect, since we are dealing with a product of the body itself.

As the changes in the intestine in passive congestion resemble grossly those in an obstruction, we took the material from the intestines of dogs upon which an Eck fistula had been done, but had failed because of the small size of the opening between the veins, and prepared it in the usual manner. Two dogs were injected with these preparations; one died with the usual symptoms, and the autopsy showed a typical picture; the other, though it was profoundly affected, recovered. A third preparation of this sort was used in the study of the lymph flow, and gave every evidence of being active.

We injected two rabbits with this toxin and obtained a reaction in each case; therefore the toxin is not specific for species, as has been found by many previous observers, working with toxins prepared by different methods.

We now proceeded to make a more detailed study of the action of the toxin on an animal. A dog was etherized and the abdomen opened; the pylorus was ligated and a cannula placed in the stomach; the intestine was ligated at the duodeno-jejunal junction, and a cannula inserted above the ligature; a cannula was tied

in the common duct; the duct of Santorini was ligated and a cannula was tied in the duct of Wirsung; a cannula was tied in the carotid and one inserted into the trachea; these last were connected with recording points on a kymograph. The ordinary appearing duodenal content filled the respective cannula; after the injection of toxin, this fluid became blood stained. The dog died; the autopsy showed the usual picture in the duodenum, down to the insertion of the cannula; beyond this point the intestine appeared normal. The contents of the stomach, of the duodenum, and of the small intestine below the ligature, were separately removed, and prepared separately in the usual manner. On injection, the stomach content produced no reaction, while that of the duodenum and of the small intestine were intensely toxic. This experiment was repeated with exactly the same result. In each case the injection of the toxin produced an abrupt fall of blood-pressure, then a gradual rise, followed by a gradual decline, until death occurred. There was no evidence of stimulation of bile flow, nor of pancreatic secretion. These experiments were instructive in that they showed the extreme rapidity with which the toxin appears in the intestinal content following injection, and in the fact that it appeared above and below the duodeno-jejunal ligature, and that it was present below the ligature even though the mucosa of this portion of the intestine showed neither hemorrhage nor even congestion.

We prepared secretion from the intestine of normal dogs. We then anæsthetized a dog and tied a cannula in the thoracic duct. The lymph was normal in appearance, and clotted rapidly. We then injected a lethal dose of toxin; the lymph became thin, blood stained, and lost its clotting power to some extent; we then injected four c.c. of secretin, and the lymph rapidly returned to normal. The dog was killed and the autopsy showed a hemorrhagic infiltration of the intestine, but not as extensive as usually noted. Another dog was prepared in the same manner, and the lymph collected; after the injection of the lethal dose, the characteristic changes in the lymph appeared. In this case we did not administer secretin, and the dog died in the course of a few hours; the autopsy showed areas of hemorrhagic infiltration of the intestine more extensive than in the first case. In neither experiment was a lymphagogic action of the toxin exhibited, which fact also tends to disprove the theory that the toxin is either a proteose or a hetero-proteose.[9, 10]

In all our work with the toxin we were greatly impressed with the rapidity with which the toxic element was excreted into the lumen of the intestine. This was shown time and again where the dogs died almost immediately after receiving a lethal dose of toxin. Autopsy was performed as soon as death occurred, and we obtained from the content sufficient toxin to kill. We feel that a justification of Sweet's criticism of Whipple's work, that the finding of the toxin in the isolated loop does not necessarily mean that it is formed there, but that it might be formed in the functionally obstructed loop, is found in the experiments in which we had ligated the intestine at the duodeno-jejunal junction, injected toxin, and were subsequently able to demonstrate toxin in both the afferent and efferent loops. These experiments also show that the toxin circulating in the blood-stream can apparently excite the cells of the intestinal mucosa to the formation of the poison, for if one considers the small amount of the toxin solution injected, and the large amount of dilution that must occur in the blood-stream and in the tissues, and the loss incident to the necessarily crude method used in recovering the

toxic element from the intestinal content, then one must conclude that it is not possible that we are dealing with the recovery of the original dose. Our experiments with the adrenalectomy and the Eck fistula dogs seem to correlate the theories dealing with disturbance of the circulation, distention and nervous irritation, and show that under such conditions a toxin can be formed; but that these conditions are not necessarily causative conditions for the formation of the poison is proven by the finding of the poison under circumstances in which exactly the opposite conditions exist, namely, lack of congestion, hyperperistalsis and extreme contraction of the gut, as was frequently seen in animals dying quickly after intravenous injection of the poison.

Our experiments with the erepsin-toxin combination show that the secretions of the lower small intestine do not exert a detoxifying action, and also that the toxin is neither a proteose nor a hetero-proteose. The lack of effect upon guinea-pigs proves that the toxin is not histamine.

A review of our work shows that a toxin is produced in high obstruction that is the same, or similar to, the toxin produced in acute pancreatitis, in acute fulminating peritonitis of non-bacterial origin, acute congestion and in paralytic ileus. This toxin may be recovered from the intestinal content, and, to a certain extent, purified. When injected intravenously, it reproduces the clinical picture of obstruction, that is, vomiting, retching, diarrhœa, tenesmus and prostration, with a subnormal temperature and a fall of blood-pressure. The autopsy findings are characteristic, and consist of more or less congestion of the mucosa of the stomach; intense congestion of the mucosa of the small intestine with hemorrhage, giving to the mucosa the appearance of purple velvet. We have occasionally found gastric ulcers, once a perforated duodenal ulcer, and once an ulcer of the cæcum. This raises the question whether the underlying cause of ulcer formation may bear some relation to this toxin. We have sometimes seen œdema of the gall-bladder and pancreas. We believe that we are justified in stating that the normal intestinal content does not contain this toxin, nor does the secretion of the lower intestine have any detoxifying properties. There is nothing in the reaction of the toxin that would lead one to believe it identical with histamine.

The results of our many and diverse lines of experimentation seem to be important in two ways; first, that they correlate and explain some of the different opinions advanced by other workers; second, they confirm the view that the site of origin is the cells of the intestinal mucosa. In considering the first point, the finding of a poison which is apparently the same as that found in high obstruction, in other conditions, tends to reconcile the contentions of writers like Murphy and Vincent [36] that the interference with the blood supply is an essential factor in the production of the toxin with their opponents; since we find it in the condition of complete portal obstruction, one must conclude that the toxin can be formed under conditions of interference with the circulation. This may also explain

the old clinical observation that a case of volvulus often runs a more severe course than does simple obstruction. On the other hand, the finding of a toxin after the removal of the adrenals, after portal congestion, and within such a short time after the intravenous injection of a fatal dose of the poison, does seem to us to rule out any essential rôle of the bacteria of the intestine; for it is inconceivable that the intravenous injection of poison, or that portal thrombosis, both of which are fatal in from a few minutes to a few hours, should in any way favor the action of the intestinal bacteria. Likewise, it is difficult to understand how adrenalectomy could change the conditions of bacterial life within the intestine.

If, as we believe, we are dealing with a disturbance of the proteolytic enzyme of the mucosa comparable entirely to the conditions existing in acute pancreatitis, in which the toxic agent is undoubtedly the tryptic ferment of the pancreas, then we can readily understand why the changes in diet could modify the final picture, as has been reported by Magnus-Alsleben,[41] Roger and Garnier,[47, 48, 49, 50] and lately by Dragstedt.[7a] We might say further that it is shown by our experiments that an injury to the mucosa or the entire wall of the intestine, which has been considered by some authors as an essential factor, is certainly not necessary.

The reason why a clinical differential diagnosis between acute pancreatitis and acute high obstruction is so difficult if not impossible, becomes clear as a result of our work; if we are correct in our assumption that the toxic element is the proteolytic ferment of the cells of the mucosa, then the two conditions are based upon a factor which is essentially the same in both instances. We therefore must expect that the general symptoms would be, as they are, precisely the same. A differential diagnosis could therefore, of necessity, be based only upon a careful study of the history of the case, such as a history of gall-stones, accidental injury to the abdomen which might have involved the pancreas, and local symptoms referable to the pancreas itself.

The finding of the same poison in the intestinal content of an animal killed by dropping into the peritoneal cavity the sterile pancreas of another dog, suggests that the two conditions may be even more closely related than a mere relationship of the toxins involved; in other words, the condition of pancreatitis may favor the production of the intestinal poison, just as does the intravenous injection of the poison itself. In this event, the two would be clinically alike because they are etiologically one and the same thing.

The explanation of the finding of the toxin after the removal of the adrenals is very difficult. It surely cannot be ascribed to a disturbance of the circulation of the intestine, as autopsy fails to reveal any such disturbance. It is possible that an, as yet unknown, control is exercised by the adrenals over the glands of the gastro-intestinal tract. Such a relationship was observed by Sweet and Pemberton;[68] these workers found that

after the removal of the adrenals, the pancreas began to secrete, the secretion becoming more and more pronounced up to the death of the animal.

The results of our experiments with secretin, while still incomplete, suggest the possibility that the course of the flow of the perverted secretion may be markedly influenced, that is, that by injecting secretin, we may produce a greater secretion of toxin into the lumen of the intestine, and consequently a lessened secretion into the lymph stream. The attempts at immunization have not offered any promise, neither in our hands nor in the reports of others in the literature. This failure of the immunization experiment might be considered to support our conclusion that we are dealing with a perverted normal product of the body, a product, which, being ordinarily normal, but under these peculiar conditions perverted, would naturally not be able to produce an immunity. At the same time, the hope that some means of defense may be found is supported by the experience which we have frequently had, and which is supported by the observations of others, that different animals of the same species react so differently to this toxin. In many instances we have observed that of two animals injected with the same dose of toxin, one would react violently and death would occur in a short time, while the other would show only a very mild and transitory reaction. Such a result can only be explained on the basis either that the second animal possessed some natural property of resistance to the toxin, or that the intravenous injection of the toxin creates some condition in the first animal that favors the production of the poison. These conditions may be subject to physiological variations; such a variation might well be produced by the character of the diet, or by the physiological condition of the animal, as in Kirstein's experiments upon the hibernating animal. We at one time believed that there was a relationship existing between the stage of digestion and the susceptibility to the toxin; but on comparing the results of experiments made upon animals recently fed with experiments on fasting animals, we were unable to arrive at any definite conclusions.

As the result of our observations we have arrived at the following conclusions:

1. That from the intestinal content in cases of high obstruction, a poison can be isolated by precipitation with alcohol, extraction with boiling water and reprecipitation with the aid of magnesium sulphate.

2. That it is not possible to obtain such a poison with this method from the intestinal content of a normal dog prepared immediately after removal.

3. That a poison which, when judged by the means at our disposal, is identical, can be obtained from conditions other than actual obstruction, such as the intravenous injection of the high obstruction toxin into normal animals, the removal of the adrenals, portal obstruction, and in experimental, acute, fulminating, non-bacterial peritonitis.

4. That this poison is undoubtedly elaborated in the cells of the greater

part of the mucosa of the small intestine, but chiefly in those of the duodenum, and that it is manifestly excreted, partly into the lumen of the intestine, but the larger part passes into the lymph stream.

5. That the clinical similarities between acute pancreatitis and high obstruction are due either to a close relationship between the toxins involved, or possibly to the fact that acute pancreatitis actually produces conditions in the intestinal mucosa favorable to the production of the same toxin as is found in cases of high obstruction.

6. Since erepsin fails to exert any action upon the toxin, and since the toxin shows no lymphagogic action whatever, it seems necessary to conclude that the toxin is neither a proteose nor a hereto-proteose.

7. That the clinical advantage of gastric lavage may be explained by the removal of the toxic content and the favoring thereby of an increased excretion into the lumen of the intestine. In addition to this treatment, should be added the introduction of large amounts of saline, both intravenously and by the rectum, to further the excretion of the toxin both by the bowel and by the kidneys.

8. That the finding of the toxin in the intestinal content after the removal of the adrenals suggests that clinically adrenalin should be added to the saline infusion in sufficient amount so that a continuous supply of adrenalin is being furnished.

BIBLIOGRAPHY

[1] Albeck: Archiv. f. klin. Chir., 1902, vol. lxv, p. 569.
[2] Amussat: Relations de la maladie de Broussais. Paris, 1839.
[3] Braun und Boruttau: Deutsche Ztschr. f. Chir., 1908, vol. xcvi, p. 544.
[4] Bouchard: Lecons sur l'autointoxications. Paris, 1887.
[5] Borszeky u. Generisch: Brun's Beitr. z. klin. Chir., 1902, vol. xxxvi, p. 448.
[6] Buchbinder: Deutsch. Ztschr. f. Chir., 1900, vol. lv, p. 458.
[7] Bokai: Archiv f. exp. Path. u. Pharm., 1888, vol. xxiv, p. 153.
[7a] Cannon, Dragstedt and Dragstedt: J. Inf. Dis., 1920, vol. xxvii, p. 139.
[8] Clairmont et Ranzi: Archiv f. klin. Chir., 1904, vol. lxxiii, p. 696.
[9] Chittenden, Mendel and Henderson: Am. J. of Phys., 1898, vol. ii, p. 142.
[10] Chittenden: Am. J. of Phys., 1898–99, vol. ii, p. 142.
[11] Cybulski und Tarchanoff: Archives internat. de Phys., 1907, vol. v, p. 257.
[12] Combe: Quoted from reference 20.
[13] Charrin: Sem. med., 1904, vol. xxiv, p. 377.
[14] Draper: J. A. M. A., 1914, vol. lxiii, p. 1079.
[15] Draper: Am. J. Med. Sci., 1909, vol. cxxxvii, p. 725.
[16] Dale: J. of Phys., 1910–11, vol. xliii, p. 318.
[17] Davis: Bull. Johns Hopkins Hosp., 1914, vol. v, p. 39.
[18] Davis and Stone: J. Exp. Med., 1917, vol. xxvi, p. 687.
[19] Dragstedt, Moorhead and Burcky: J. Exp. Med., 1917, vol. xxv, p. 421.
[20] Enderlein und Hotz: Mitt. a. d. Grenzgeb. d Med., u. Chir., 1911, vol. xxiii, p. 755.
[21] Esau: Archiv f. klin. Chir., 1910, vol. xcii, p. 155.
[22] Esau: Beitr. z. klin. Chir. 1908, vol. lx, p. 508.
[24] Gley et LeBas: Archives de Phys., 1892, p. 848.
[25] Hartwell and Hoguet: J. A. M. A., 1912, vol. lix, p. 82.
[11] Hartwell and Hoguet: Am. J. Med. Sci., 1912, vol. cxliii, p. 357.

[38] Humbert: Quoted from reference 20.
[39] Helmburger und Martini: D. Ztschr. f. Chir., 1904, vol. lxxiv, p. 527.
[40] Kocher: D. Zeitschr. f. Chir., 1877.
[41] Krafft: Quoted from reference 20.
[42] Kukula: Archiv f. klin. Chir., 1901, vol. lxiii, p. 773.
[43] Kukula: Archiv f. klin. Chir., 1901, vol. lxv, p. 773.
[44] Kirstein: D. med. Wchnschr., 1899, p. 1000.
[45] Kader: D. Ztschr. f. Chir., 1892, p. 33.
[46] Murphy and Vincent: Boston Med. and Surg. J., 1911, vol. clxv, p. 684.
[47] Murphy and Brooks: Arch. Int. Med., 1915, vol. xv, p. 392.
[48] McKenna: Surg., Gyn. and Obst., 1913, vol. xvii, p. 674.
[49] McLean and Andries: J. A. M. A., 1912, vol. lix, p. 1614.
[50] Maury: J. A. M. A., 1907, 19/2,1003, 1910, vol. i, p. 5.
[51] Magnus-Alsleben: Hoffmeister's Beitr., 1904–5, vol. vi, p. 503.
[52] Nicholaysen: Quoted from reference 20.
[53] Nesbitt: J. Exp. Med., 1899, vol. iv, p. 1.
[54] Nothnagel: Z. f. klin. Med., 1882, vol. iv, p. 223.
[55] Nothnagel: Virchow's Archiv, Bd. vol. xxvi, p. 88.
[56] Roger et Garnier: Compt. Rend., 1905, vol. lix, pp. 388, 674, 677.
[57] Roger et Garnier: Compt. Rend., vol. lxiv, pp. 426, 610, 83.
[58] Roger et Garnier: Compt. Rend., 1908, vol. lxv, pp. 202, 389.
[59] Roger et Garnier: Rev. de Med., 1906, vol. xxvi, p. 953.
[60] Richardson: Boston Med. and Surg. J., 1920, pp. 183, 10, 288.
[61] Reichel: D. Ztschr. f. Chir. 1892-3, vol. xxxv, p. 495.
[62] Reichel: Quoted from reference 20.
[62a] Sigfried: Abdelhalden's Handbuch, 1911, vol. iv, p. 198.
[63] Stone, Bernheim and Whipple: ANNALS OF SURGERY, 1914, vol. lix, pp. 1, 714.
[64] Stone, Bernheim and Whipple: J. Exp. Med., 1914, vol. xix, p. 144.
[65] Stone, Bernheim and Whipple: J. Exp. Med., 1913, vol. xvii, pp. 286, 307.
[66] Stone, Bernheim and Whipple: Bull. J. Hopkins Hosp., 1912, vol. xxiii, p. 159.
[67] Sweet: Penna. Med. J., April, 1913, 1916.
[68] Sweet, Peet and Hendrix: ANNALS OF SURGERY, June, 1916.
[68a] Sweet and Pemberton: Arch. Int. Med., 1910.
[69] Sauerbruch und Heyde: Z. f. exp. Path. u. Therap., 1909, vol. vi, p. 34.
[70] Talma: Ztschr. f. klin. Med., 1890.
[71] von Jaksch: Wein. klin. Wchschr., 1892, p. 52.
[72] von Khautz: Arch. f. klin. Chir., 1908-09, vol. lxxxviii, p. 42.
[73] Wilms: Deutsche Chirurgie Lieferung, vol. lxxviii, p. 46g
[74] Wrzosck: Virchow's Archiv, 1904, vol. lxxviii, p. 82.
[75] Wells and Osborne: J. of Infect. Dis., 1915, vol. xvii, p. 259.
[76] Whipple, Rodenbaugh and Kilgore: J. Exp. Med., 1916, vol. xxiii, p. 123.
[76] Whipple: Cont. to Med. and Biol. Research, July 12.
[79] Zunz: Abderhalden's Handbuch, 1911, vol. v, p. 610.
[71] Guillaume: Presse Médicale, 1921, p. 84, October 19.
[72] Bacon, Anslow and Eppler: Archives of Surgery, 1921, vol. iii, p. 641.
[73] Stone: Surgery, Gyn. and Obst., 1921, vol. xxxii, p. 415.

THE PRESENT STATUS OF EPIPLOPEXY*

WITH THE REPORT OF TEN CASES

By John H. Gibbon, M.D.

and

John B. Flick, M.D.

of Philadelphia

The surgical measures employed for the relief of cirrhosis of the liver have been various, but all directed at the diversion to the systemic circulation of a part of the venous blood going to this organ and carrying poisonous products, which the liver should normally eliminate. The idea of bringing relief to the liver by this diversion of the venous blood was conceived, like so many new ideas in medicine, independently and at about the same time by several men. The surgical procedure first suggested and practised consisted in irritating by gauze friction the upper surfaces of the liver and spleen and the under surface of the diaphragm, and the fixation of the omentum to or in the abdominal wall. Talma, of Utrecht, suggested this operation in 1889 and the first three operations were done by Dutch surgeons, von der Meule (1889), Schelkly (1891), and Lens (1892). The last patient survived the operation six months, the first two died shortly after operation.[1] Rutherford Morison, of New Castle, at the suggestion of Drummond, who knew nothing of Talma's suggestion or of the work of the Dutch surgeons across the channel, performed the first successful operation in 1894 and 1895. These cases were reported in 1896.[2] Talma's first paper appeared in 1898.[3]

Owing to the publications of these three men the operation has been designated by subsequent writers as the "Talma," or the "Talma-Morison," or the "Talma-Morison-Drummond" operation, though they called it omentopexy or epiplopexy, as the fixation of the omentum was considered the important step in the procedure. It is with the present status of this operation that we will deal, but it is well first to consider briefly the other surgical means suggested or practised for the relief of hepatic cirrhosis.

The idea of anastomosing the portal vein and the vena cava, the establishment of the so-called "Eck's fistula," would at first sight seem a rational one, provided of course the operation carried with it little risk; but notwithstanding the accomplishment of such a fistula by Vidal and several other surgeons,[4] the operation has proved too dangerous a one and no patients have survived long enough to enable us to form any opinion as to its effect on the overburdened liver.

The ligation of some of the main tributaries of the portal vein has been done. Mayo[5] speaks of the ligation of the inferior mesenteric or superior rectal vessels as a supplement to epiplopexy and Moynihan[6] mentions ligation of the inferior mesenteric vein as an additional means of diverting blood from

* Read before a joint meeting of the New York Surgical Society and the Philadelphia Academy of Surgery, November 7, 1921.

the portal circulation. Ligation of the splenic artery with the object of producing atrophy of the spleen has been suggested by J. Gerster and Hartmann and others have reported cases, but the mortality has been large (Moynihan).

Anastomosis of the superior mesenteric and spermatic or ovarian veins, although accomplished many times, has not been successful.

Transplantation of the testicle and the spermatic cord to the abdomen and surrounding it with the omentum was recommended and practised by Lanz, 1911, and Lorenz reported two cases well two years after the performance of this operation.

All kinds of drainage of the ascitic fluid have been tried. Repeated aspirations have cured some cases, probably due to the resulting adhesions. External drainage, notwithstanding its advocacy by so excellent a surgeon as Rutherford Morison, has not appealed to most surgeons, because of the danger of infection of the peritoneum. Our own plan has been always to take pains to prevent leakage of the fluid after operation. Subcutaneous drainage of the fluid by means of the implantation of silk threads (Lambotte), sections of blood-vessels, glass and rubber tubes, has not been successful after extensive trial. Handley has employed the femoral canal for subcutaneous drainage, but excepting one "brilliant result," the operation has proved unsatisfactory, as the new canal connecting the peritoneum and the connective tissue of the thigh becomes obliterated (Binnie). Routte, in 1907, first tried anastomosing the saphenous vein with the peritoneum and the operation seemed to offer some chance of successful drainage of the ascitic fluid, but subsequent reports by several surgeons go to show that the drainage is only temporary, as the vein becomes occluded or obliterated.[7]

In 1891 Terrier performed cholecystostomy and maintained the biliary fistula, urging its use for the cure of cirrhosis, on the ground that this condition resulted from infection from the intestinal tract through the bile ducts. This operation was later popularized in France, largely by the publication of many successes by Delangeniére. Greenough,[8] in 1902, collected seventeen cases, nearly all French, of which thirteen were relieved. This operation, however, does not seem to have been given much attention by American or English surgeons. Cholecystenterostomy has also been done in a number of cases. It would seem that both cholecystostomy and cholecystenterostomy could only relieve those cases of cirrhosis due to infection through the bile ducts and gall-bladder.

Direct drainage of the liver by puncture through the abdominal wall, done with the idea of relieving the engorged liver, is not an operation which appeals to surgeons.

A number of successes have apparently followed visceropexy and a few the simple opening and closing of the abdomen, as is done in the moist type of tuberculous peritonitis. Any successes following these measures must be due to the resulting adhesions and tend to confirm the reasoning of Talma and Morison.

Splenectomy, where an enlarged spleen accompanies hepatic cirrhosis, is recommended very strongly by W. J. Mayo. He has done this operation in six cases and five were greatly relieved. In splenic anæmia he has found cirrhosis of the liver in a considerable number of cases and here the splenectomy seemed to cure the cirrhosis. Splenic enlargement in cirrhosis of the liver with ascites does not seem to have been a common condition, but should it be found, splenectomy would seem to be indicated, in view of Mayo's results. Certainly the removal of the spleen cuts down to an enormous degree the blood passing to the portal vein and both Mayo and Moynihan refer to the poisons carried from the spleen to the liver by the venous channels and these of course are eliminated by splenectomy.

Epiplopexy.—Since the early publications of Morison and Talma, many contributions to this subject have been made from many countries and various modifications of the original technic have been suggested. Among the early notable papers in this country was one by Packard and Le Conte (*Amer. Jour. Med. Sciences,* 1901), including the report of two cases: another by Greenough (*Amer. Jour. Med. Sciences,* 1902), who presents a summary of 105 cases collected from the literature. The mortality within thirty days in these cases was twenty-nine and one-half per cent., and there was improvement in forty-two per cent. M. L. Harris [9] analyzed the reported cases and several of his own and took a rather pessimistic view of the results obtained. He did not agree with the reasoning of Talma, but believed that chronic inflammatory changes in the peritoneum were influential in the production of ascites. White [10] and other Englishmen hold the same opinion.

Many valuable papers have been presented during the past twenty years and show very different views as to the benefit to be derived from epiplopexy.

Morison [11] reports one case well for seven years after operation and another who lived for six years, dying then following an operation for ventral hernia, and a third died two years after operation from pneumonia. W. J. Mayo [12] has performed epiplopexy twenty-eight times. There were four deaths following the operation and eight patients died later. The remaining sixteen patients he reports as relieved. Moynihan has operated seven times, one case being cured, another relieved. The literature contains many reports of one or two cases which have remained well for a number of years after operation. Large collections of cases have been made by Montprofit [13] in France, by Landenburger [14] in Germany and White [10] in England. Eliot and Colp [15] present a study of twenty-three cases operated upon in the Presbyterian Hospital in New York out of 127 cases of cirrhosis of the liver admitted to the hospital during the previous seventeen years. In eighteen cases the cirrhosis was due to the excessive use of alcohol, two were syphilitic and in two the ascites was associated with an enlarged liver due to cardiovascular disease. One of these last two died directly after operation, but the other is living and greatly relieved three years after operation. There were eight post-operative deaths in the twenty-three cases. The end results were

known in but seven of the remaining fifteen cases and these had all derived benefit from the operation and were living from three months to three years after it. Riesman [16] takes an optimistic view of the condition and urges further use of epiplopexy. He says that "cirrhosis of the liver is one of the few chronic non-bacterial visceral diseases, perhaps the only one, that may actually be cured." He states that if the interval between the tappings for ascites is decreasing, tapping should be discontinued and the patient operated upon.

One is struck in reviewing the literature with the fact that some cases are apparently cured, while others, the greater number, are not benefited at all. It was with the hope of trying to arrive at some explanation of this fact that we took up the study of our own cases, ten in number, and compared them with those already reported.

While our results have not been strikingly good, they have been good enough to make us feel that the operation of epiplopexy is a useful one and one which we can expect to result in cure in probably ten per cent. of the cases and in benefit to a much larger percentage.

W. J. Mayo puts the cirrhoses of the liver into two main groups, one he calls "portal cirrhosis," and the other "biliary cirrhosis." (1) "Portal cirrhosis, in which the irritants, bacterial, toxic, and biochemical substances are received from the intestinal tract and from the spleen by the way of the portal vein, and in which the connective tissue is deposited about the radicles of the portal vein. (2) Biliary cirrhosis, in which the infectious agents reach the biliary ducts by extension of infections from the gall-bladder, and great bile ducts, or from hæmatogenous infections commonly portal or, not rarely systemic, such as those following pneumonia, typhoid, focal lesions, etc., and in which the connective tissue deposit is related to the biliary ducts. The many varieties of cirrhosis described are to be looked on as variations and combinations of these two main divisions."

"Portal cirrhosis when advanced gives rise to the clinical symptoms which depend on portal circulatory obstructions shown by ascites and gastric hemorrhages. Jaundice is absent or a terminal condition. Local areas of portal cirrhosis are not infrequent and often without symptoms."

"Biliary cirrhosis, on the contrary, depends on infections in the biliary ducts, and if the whole or greater part of the liver is involved, jaundice is an early, prominent and continuous feature. Ascites is absent or a terminal manifestation. It would appear that we ordinarily recognize as biliary cirrhosis only the late stages of a relatively frequent liver condition after more or less permanent and extensive damage to the liver tissues has taken place. Infections of the gall-bladder and biliary tract are often accompanied by localized cirrhotic processes without definite symptoms."

Whether or not one accepts this classification, it is well to bear in mind the different sources from which cirrhosis may come, and we think it explains to some extent some of the successes and failures of the different operations. For instance in the "portal" type, biliary drainage, except as

the operation produces adhesions, could be expected to do little good, and in the " biliary " type epiplopexy could hardly produce a cure.

It seems quite evident that the alcoholic cases are the ones giving the best results and the syphilitic the worst. Morison says that we should be able to cure by epiplopexy every case of alcoholic cirrhosis. The majority of our own cases were not alcoholics. He thinks that no syphilitic case is cured by the operation. One of our syphilitic cases, who has survived the operation five and one-half years, has still to be tapped at regular intervals.

Greenough, from the study of his collection of cases, concluded that the hypertrophic type gave better results than the atrophic, and White and Eliot's collections tend to confirm this fact. In all of our ten cases the liver was contracted and in but one was enlargement of the spleen noted. The results in the cases operated upon early in the disease have been better than where the operation was postponed for a long time. Eliot reports a case, however, of marked cardiovascular disease which was benefited and one of our cases presented the same condition. The cases in which ascites is of slow development give better results than those in which it takes place rapidly.

The association of tuberculous peritonitis and cirrhosis of the liver is an interesting one and probably explains to some extent the difference in opinion as to the source of the ascites. Hertzler [17] states that cirrhosis of the liver occurs in about twelve per cent. of the cases of tuberculous peritonitis. In one of our cases I found the omentum rolled up and fixed and was unable to do anything but open and close the abdomen. This patient was tapped eighteen times before operation, and of course the condition found may have been due to the previous tappings. He is living, nearly a year after operation, and has been tapped once since. I think it is quite possible that this is a case of tuberculous peritonitis.

Operative Technic.—Since there seems to be no doubt that extensive adhesions produce the desired results and that too extensive operative procedures are the cause of a rather high mortality, it would appear that we should make the operation as short and simple as possible. Our own experience has taught us to eliminate elaborate technic and prolonged anæsthesia, and therefore in our recent cases we have simply fixed the omentum to the parietal peritoneum by two rows of mattress sutures passed through the recti about two inches to either side of the midline, the skin being reflected to permit the necessary exposure of the muscles. This requires but a very few minutes. We have used ether, chlorid of ethyl, gas-oxygen and infiltration anæsthesia, all preceded by morphia, and we believe that the gas-oxygen is probably the best.

Our operative mortality was two out of the ten cases, one died of pneumonia forty-eight hours after operation and the second four days after operation from peritonitis. These deaths occurred when we were following the more elaborate procedure. In the last six cases there has been but one death with which the operation might be associated. This patient died two months after operation; she was a syphilitic.

Two modifications of the Morison technic have been extensively employed. Schiassi placed the omentum between the peritoneum and the abdominal muscles, and Narath brought it out between the recti muscles and fixed it under the skin.

Although these operations apparently have given good results, I think that both add to the operative risk. The same may be said of Morison's method in which drainage of the pelvis by a glass tube is employed. Of course this would seem also to increase the risk of subsequent infection. We think that most operators have eliminated friction of the liver, spleen and diaphragm because they believe it is productive of shock. In Greenough's collection of cases, the Talma-Morison operation was accompanied by a mortality of thirty-two per cent. and the Schiassi operation by only seventeen per cent. It must be remembered, however, that in the Talma-Morison cases, friction of the viscera and drainage of the pelvis were probably employed.

We have had no experience with ligation of the mesenteric veins, as an additional means of cutting off the supply of portal blood. It must be remembered that in the majority of cured cases, aspirations of the fluid subsequent to operation have been necessary. The reaccumulation of fluid does not mean that the operation is a failure unless it is persistent.

The following is a summary of our ten cases, all of which have been followed:

One case died forty-eight hours after operation from lobar pneumonia (Case III).

One case died four days after operation from peritonitis; operation under infiltration anæsthesia followed by wound infection (Case IV).

One case died two months after operation, apparently from toxæmia (Case VII).

One case died about three months (?) after operation at another hospital; cause of death unknown (Case I).

One case relieved of all symptoms for three years, then died of apoplexy (Case II).

One patient relieved of symptoms for eighteen months and able to work, then had several profuse gastric hemorrhages, which were controlled under rest, and the patient discharged from the hospital in good condition. No ascites. Died four years after operation; cause of death unknown (Case V).

One case is alive eighteen months after operation and in good health; has gained twenty-five pounds in weight. No evidence of a reaccumulation of fluid (Case VIII).

One patient is alive six months after operation and able to do light work. No reaccumulation of fluid since discharge from hospital (Case X).

One case, a syphilitic, is alive five years after operation with no relief of symptoms (Case VI).

SYNOPSIS OF CASES

CASE I.—Pennsylvania Hospital, No. 1416; operation, September 6, 1910. S. C., white, female, aged forty-seven years; no alcoholic or syphilitic history.

No jaundice, no marked dilatation of superficial veins of abdominal wall, no gastric symptoms, no history of hemorrhage. Œdema of lower extremities for four or five months before operation. Urine ex. specific gravity 1030, heavy cloud of albumin. No pre-operative tappings. Duration of symptoms one year. Operation ten weeks after enlargement of abdomen was first noticed. Morphia-ether anæsthesia. The liver at operation was contracted and hard; the veins of the round ligament were greatly distended; there was no enlargement of the spleen; there were no adhesions present. Omentum attached to the abdominal wall to left of incision, peritoneum separated from the muscles on the right and a large mass of omentum inserted into cavity thus produced, and the upper surface of the liver and the under surface of the diaphragm scrubbed with gauze. Abdomen closed without drainage. Reaccumulation of fluid occurred, and on the third day a small opening was made through the wound for the purpose of drainage. Six weeks after operation abdomen was tapped and 6000 c.c. of fluid removed. Patient left the hospital against advice and died a few months after operation at another hospital.

Case II.—Jefferson Hospital, B426; operation, July 18, 1911. M. Z., white, female, aged sixty years; no alcoholic or syphilitic history, but history of attacks of pain which seemed like gall-stone colic and often followed by jaundice years ago. Has not suffered from these attacks for the past three or four years. Loss of weight; vomiting at irregular intervals; no hemorrhage; no œdema of extremities; no dilatation of superficial veins of abdomen. Urine ex. sp. gr. 1017, faint trace of albumin. Blood ex. hæmoglobin seventy per cent.; red blood-cells, 5,100,000; white blood-cells, 7600. One pre-operative tapping was done a year after enlargement of abdomen was first noticed. Operation under morphia-chlorid of ethyl-ether anæsthesia. Liver was contracted and nodular throughout. No enlargement of spleen. There were numerous adhesions about the liver; the colon was adherent so that the gall-bladder could not be felt. The omentum was fixed to the parietal peritoneum on the left side and a considerable portion of the omentum fixed in a pocket made by dissecting up the peritoneum on the right side. Abdomen closed without drainage. There was a post-operative reaccumulation of fluid which disappeared without tapping. Patient discharged from hospital one month after operation and was relieved of symptoms for two years; then died of apoplexy. She never suffered a recurrence of ascites.

Case III.—Pennsylvania Hospital, No. 1941; operation, October 22, 1913. A. T., white, male, aged sixty-one years, moderate drinker of beer, Wassermann negative, duration of symptoms eight weeks. At the onset of illness abdomen painful, had a chill, some vomiting and was slightly jaundiced. No dilatation of superficial veins of abdominal wall; no œdema of lower extremities; no hemorrhage. Urine ex. sp. gr. 1030, trace of albumin; few leucocytes and hyaline casts. Leucocyte count 6900. First tapping about three weeks after onset of symptoms, 5715 c.c. of fluid removed. There were four pre-operative tappings and between five and six thousand c.c. of fluid removed each time. Operation under morphia-chlorid of ethyl-ether anæsthesia. Liver was contracted to about one-fourth its normal size and the veins of the omentum were enlarged. No enlargement of the spleen. No adhesions. The omentum was split and the two halves crossed and each inserted into a pocket on either side of the wound made by dissecting up the peritoneum. Abdomen closed without drainage. Death forty-eight hours after operation from lobar pneumonia.

Case IV.—Pennsylvania Hospital, No. 2699; operation, November 12, 1913. D. D. G., white, male, aged forty years, used alcoholic beverages to excess. chancre at seventeen years (Wassermann not recorded). History of attack of pain in right hypochondriac region, necessitating morphia, two years previous. Second attack similar in character, but on left side, six months later. No jaun-

dice. During present illness slight loss of weight; discomfort in epigastrium; œdema of legs; hemorrhoids; no dilatation of the superficial veins of abdomen. Urine ex. sp. gr. 1016, faint trace of albumin, few granular casts. No pre-operative tappings. Operation four weeks after enlargement of abdomen was first noticed under infiltration anæsthesia. Liver contracted and hard; no enlargement of spleen; no adhesions. The omentum was divided, the two halves crossed and each inserted into a subperitoneal pocket. Death four days after operation from peritonitis.

Case V.—Jefferson Hospital, D4726; operation, February 28, 1914. A. M., white, male, aged fifty-two years, heavy drinker of beer, no history of syphilis. Duration of symptoms about one year. Epigastric pain, worse during day, not influenced by the taking of food. Bowels irregular. Has had several profuse gastric hemorrhages, is pale and has lost between twenty and thirty pounds in weight in the past year. Sclera icteroid; no dilatation of superficial veins of abdomen; no œdema of extremities; no free fluid demonstrable before operation. Urine examination negative. Fæces negative for occult blood. Blood examination, hæmoglobin seventy-two per cent.; red blood-cells, 4,120,000; white blood-cells, 8800. It was a question whether this patient had a cirrhosis of the liver or a malignant growth. Operation under morphia-chlorid of ethyl-ether anæsthesia. Liver found contracted and irregular, considerable free fluid, no enlargement of spleen, no adhesions. Omentum attached to abdominal wall by three mattress sutures on each side of the incision and wound closed. Post-operative course uneventful. Discharged from hospital five weeks after operation. Patient well of symptoms and working for eighteen months, then several profuse gastric hemorrhages, controlled by rest, and the patient discharged from the hospital in good condition. No ascites. Died four years after operation; cause of death unknown.

Case VI.—Bryn Mawr Hospital; operation, June 9, 1916. H. M., white, female, aged sixty years, history of intemperate life, Wassermann strongly positive. It was a question whether this patient had a cirrhosis of the liver or a malignant growth of the colon. There was considerable fluid in the abdomen, but the X-rays suggested some obstruction at the hepatic flexure. Operation under morphia-gas-oxygen anæsthesia. There was marked cirrhosis of the liver and an enormous quantity of fluid in the abdomen; no enlargement of the spleen; no adhesions. The omentum was sutured in a pocket between the peritoneum and muscle. Post-operative course uneventful, but there was a reaccumulation of fluid. Five years later patient is alive and able to do light work, but has never been free from ascites. She has had to be tapped at intervals of a few weeks ever since her discharge from the hospital. She now has marked dilatation of the superficial veins of the abdominal wall.

Case VII.—Pennsylvania Hospital, No. 4280; operation, January 17, 1917. M. I., white, female, aged fifty-three years, no history of alcohol, Wassermann strongly positive. Cough, night sweats and loss of weight for the past year; abdomen noticeably swollen for the past four months. Past few months dyspnœa; nausea at times, but no vomiting; no jaundice, no hemorrhages; no œdema of lower extremities; no dilatation of superficial veins of abdomen. Urine examination, sp. gr. 1020, light cloud of albumin, few hyaline and granular casts. No pre-operative tappings. Operation four months after swelling of abdomen was first noticed under morphia-chlorid of ethyl-ether anæthesia. Liver rough and contracted; no enlargement of spleen; no adhesions, except about uterus, which contained fibroids. Omentum sutured to incision and abdomen closed. A week after operation there was some bleeding from the wound, controlled by packing. There was a reaccumulation of fluid. Five weeks after operation

abdomen was tapped and 8200 c.c. of fluid removed. Eight weeks after operation evidence of toxæmia developed, patient became jaundiced and died.

Case VIII.—Jefferson Hospital, I6316; operation, April 26, 1920. E. O., white, male, aged thirty-three years, no history of the use of alcohol; Wassermann negative. Previous operation four months ago in a New York hospital, apparently for an ascites, six weeks after swelling of abdomen was first noticed. Tapped three times before first operation and about fifteen times between first and second operations. Patient states that he has lost weight; no gastric symptoms; no jaundice; no œdema of lower extremities; no hemorrhage. Superficial veins prominent over entire abdomen. Urine examination, sp. gr. 1024, faint trace of albumin. Blood examination, hæmoglobin eighty-nine per cent., red blood-cells 4,550,000, and white blood-cells 14,000. Operation under morphia-gas-oxygen anæsthesia. Liver contracted and studded with small nodules; no evidence of tuberculosis; no enlargement of spleen; omentum not adherent to old scar, but adherent to parietal peritoneum on the left side. It was fixed about the wound to the parietal peritoneum over an area of about two and one-half by three and one-half inches by stitches passed through the muscle. There were ten post-operative tappings; the first about two weeks, and the last about three months after operation. Eighteen months after operation patient is in good health; has gained twenty-five pounds in weight and there is no evidence of free fluid in the abdomen.

Case IX.—Jefferson Hospital, No. J4634; operation, February 15, 1921. J. E., white, male, aged thirty-eight years, no history of alcohol, Wassermann negative. Duration of symptoms eight months; cough, dyspnœa; loss of weight; œdema of lower extremities; no dilatation of superficial veins of abdomen; no jaundice; no hemorrhage. Urine examination, sp. gr. 1025, faint trace of albumin, occasional hyaline casts. Blood examination, hæmoglobin eighty-three per cent.; red blood-cells 4,400,000; white blood-cells 9600. Sputum examination negative for tubercle bacilli (five examinations). First tapping less than two months after patient first noticed swelling of abdomen. Tapped eighteen times before operation. Operation under morphia-gas-oxygen anæsthesia. Liver was contracted and nodular and adherent to the abdominal wall, the only place that it was free being far around on the right lobe. Numerous recent adhesions. Omentum appeared to be rolled up anl adherent across the abdomen. Exploration only; abdomen closed. Reaccumulation of fluid after operation, but patient was discharged from hospital three weeks after operation in fair general condition. Eight months after operation family physician writes that patient is living, but that abdomen is still distended. He was tapped once but only a quart of fluid obtained. Present condition is poor.

Case X.—Pennsylvania Hospital, No. 990; operation, April 27, 1921. N. F., white, male, aged fifty-one years, history of moderate use of beer and wine, Wassermann negative. Duration of symptoms two months; cough; loss of weight; enlargement of abdomen; no jaundice; no œdema of lower extremities; no hemorrhages; no dilatation of superficial veins of abdominal wall. One preoperative tapping six weeks after swelling of abdomen was first noticed, 1500 c.c. of fluid removed. Operation under morphia-gas-oxygen-ether anæsthesia. Liver contracted and hob-nailed; gall-bladder tense and could not be emptied by pressure; no stones felt; head of the pancreas very hard; spleen enlarged to about three times its normal size; no adhesions. Omentum attached to abdominal wall by four mattress sutures on each side of incision. Post-operative course uneventful. Fluid reaccumulated, necessitating tapping once, this shortly before discharge from the hospital, nineteen days after operation. Patient alive six months after operation and able to do light work. No reaccumulation of fluid since discharge from hospital. Spleen still enlarged. Blood examination,

hæmoglobin eighty per cent.; red blood-cells 3,760,000; white blood-cells 6900. Red blood-cells normal in size and shape; no degeneration.

REFERENCES

[1] Packard and LeConte: Amer. Jour. of the Med. Sciences, March, 1901.
[2] Drummond and Morison: Brit. Med. Jour., September 19, 1896.
[3] Talma: Berl. klin. Wchnschr., September, 1898.
[4] Jos. Ransohoff and J. Louis Ransohoff: Surg. Diag. and Treat., Oschner, vol. xi, Lea and Febiger, 1921.
[5] W. J. Mayo: Collected Papers of the Mayo Clinic, vol. x, 1918, W. B. Saunders & Co.
[6] Sir Berkeley Moynihan: The Spleen and Some of Its Diseases. W. B. Saunders & Co., 1921.
[7] Binnie: Manual of Operative Surgery. P. Blakiston's Son and Co., 1921.
[8] Greenough: Amer. Jour. Med. Sciences, December, 1902.
[9] M. L. Harris: The Chicago Med. Recorder, August, 1902.
[10] Sinclair White: Brit. Med. Jour., November 10, 1906.
[11] Morison: Brit. Med. Jour., January 20, 1912.
[12] W. J. Mayo: ANNALS OF SURGERY, vol. lxviii, 1918.
[13] Montprofit: French Surg. Cong., 1904.
[14] Ladenburger: Die Talmische Operation. Wuerzburg, lxviii, No. 7.
[15] Eliot and Colp: Surg., Gyn. and Obst., vol. xxviii, 1919.
[16] Divid Riesman: Jour. A. M. A., January 29, 1921.
[17] Hertzler: The Peritoneum, vol. xi. C. V. Mosley Co.

BLEEDING ULCER OF THE DUODENUM ASSOCIATED WITH CHOLECYSTITIS*

By Edward Starr Judd, M.D.
of Rochester, Minnesota
section on surgery of the mayo clinic

Within the past year I have operated on four patients with bleeding duodenal ulcers in whom the pathologic condition was more extensive in the gall-bladder than in the duodenum. In each case the duodenal ulcer could be demonstrated easily and in one case it was of long standing, as evidenced by the amount of scar tissue. A very severe grade of cholecystitis was also present in all of these cases. The gall-bladders of the four patients were very much alike, being rather larger than normal, with thick, œdematous They were not compressible, because of the inflammatory deposits in the tissues. They contained stones and infected bile in each instance. I believe that their mucous membranes were completely destroyed and that they were functionless.

These cases of cholecystitis and duodenal ulcer were also very similar clinically. The chief symptom in each case was gastro-intestinal hemorrhage, usually very severe, occurring at intervals of a few months. Two of the patients arrived for treatment just after hemorrhage; one had had a severe hemorrhage on the train the night before. The two patients had a hæmoglobin of about thirty per cent. on first examination. All four patients complained of mild dyspepsia which was easily controlled by regulation of the diet and proper management. The massive hemorrhages occurred when least expected, often when the patients had been symptom-free for a long time. None of the four had ever had severe pain. It was impossible to elicit a history of gall-stone colic or any other symptom suggestive of a disease of the gall-bladder. In one patient, an elderly man who had had very severe hemorrhages at intervals for many years, and who had a great deal of scar tissue in the wall of the duodenum, the lumen had been greatly reduced. Following a gastro-enterostomy some years previously, he had been relieved for a time, but the attacks of hemorrhage returned. A severe hemorrhage occurred just before his arrival at the Clinic, and operation was postponed for several weeks in order to allow him to recover. He had marked arteriosclerosis which may have accounted for the apparent ease of the bleeding. Four weeks later when the operation was performed there was no evidence of a break in the mucous membrane of the duodenum or of vessel erosion as possible sources of bleeding. There was a great deal of scar tissue in the wall of the duodenum, inflammation in the tissues of the gall-bladder, and a definite inflammatory process throughout the liver. Hepatitis had progressed almost to the stage of cirrhosis. There was no jaundice or ascites.

* Read before the Southern Surgical Association, December, 1921.

The findings at operation on the other three patients differed somewhat. There was definite ulceration on the anterior part of the duodenum away from the larger vessels which appeared quite unimportant. There was no induration or deformity of the duodenal lumen. It is possible that there were other and deeper ulcers which were not found, but I believe that these small areas of duodenitis in the region of the duodenal cap were the only lesions in in the duodenum. There was extensive hepatitis and cholecystitis, with stones and infected bile in the gall-bladder. While tissue was not removed from the liver for microscopic examination, we nevertheless were convinced that the hepatitis was the type usually seen in association with cholecystitis and that it had not reached the stage of cirrhosis.

The findings in these four patients impressed us with the importance of infections in the gall-bladder as a possible etiologic factor in cases of gastro-intestinal bleeding. A definite lesion in the duodenum was found in every instance and undoubtedly was the point from which bleeding occurred. The hemorrhages were of the massive type, such as usually occur from the pancreatico-duodenal artery, and yet in each case it was quite definitely shown that none of the larger vessels could be involved in the ulcerations.

There is sufficient evidence to show that gastro-intestinal bleeding may occur as a result of infection in the gall-bladder and liver. Balfour mentions a case in which bleeding ceased after the removal of a chronically inflamed gall-bladder. Deaver reports profuse bleeding in hemorrhagic infections of the biliary tract. In one of his cases blood reached the duodenum through the common duct and then regurgitated to the stomach; the primary cause was streptococcic cholecystitis. Kelling, in an article on the relation between cholelithiasis and ulcer of the duodenum, in speaking of the differential diagnosis between gall-bladder disease and ulcer of the duodenum, says that occult blood with duodenal ulcer may mean nothing; it may also come from infections in the gall-bladder.

Some months ago I operated on a patient in whom hemorrhage occurred regularly every few days. Ulcer could not be found, but cholecystitis with stones, and extensive scarring and œdema of the liver, which oozed a great deal as the gall-bladder was removed, were noted. The history of this patient is as follows:

CASE I (A374792).—Mrs. W. R. A., aged fifty-three years, came to the Clinic October 31, 1921, because of gastric hemorrhage. The history of trouble began nineteen years before with indigestion and upper abdominal cramps. The pain passed "through and through" the right hypochondrium and lasted two or three hours. Relief was obtained by soda or by hypodermic injections of morphin. After the pain had subsided the entire abdomen was tender. The patient had never been jaundiced. About ten years before she began to have attacks of gas and distress with belching two or three hours after meals; this was aggravated by certain foods, and was relieved by soda. In the past three years the distress had increased and usually commenced about one hour after meals. She was often wakened at night by the pain. After eating apples a few months before she was awakened by severe, acute indigestion

which required a hypodermic. The next day she noticed black stool, and vomited small amounts of coffee-ground material. Shortly after this she was treated for ulcer and was pronounced cured. One month later she had a second gastro-intestinal hemorrhage, and since that time bleeding spells occurred at regular intervals. She had lost thirty pounds in weight.

Examination revealed systolic blood-pressure 135 and diastolic 85. The hæmoglobin was sixty per cent. Röntgen-ray examination of the gastro-intestinal tract was negative.

Operation was performed November 19, 1921. Hepatitis was very marked, the liver being almost cirrhotic. There was also chronic cholecystitis with stones, inflammation of the appendix, and œdema in the tissues at the pyloric end of the stomach. The ascending colon was bound down in a manner to suggest an old abscess. It was thought possible there might be a lesion causing the intestinal bleeding, but none was found. The gall-bladder and appendix were removed. The patient vomited a little bloody material on one or two occasions after the operation, but very shortly signs of improvement were noted. Although the time since operation is short, the patient is gaining and apparently is in better condition than she had been for a long time. Bleeding has entirely stopped.

Several years ago Crispin in the Clinic studied a series of cases of gall-bladder disease and found a history of gastro-intestinal bleeding in about five per cent. Communication with these patients revealed, in nearly every instance, that after the gall-bladder had been attended to bleeding ceased. In a recent review of all the cases of gall-bladder disease observed in the Clinic in 1918 and 1919, we found that hemorrhage was mentioned as a symptom in 2.43 per cent.

Rankin has recently reviewed the histories of fifty-five patients in the Clinic with blood in the vomitus, in the stool, or in both, but in whom a pathologic condition was found in the gall-bladder at operation. The average age of these patients was thirty-nine and one-half years, the oldest being sixty-seven and the youngest eighteen. There were thirty-six females and nineteen males, a ratio of almost two to one. In the majority of males diagnoses had been made of gastric or duodenal ulcer, but in many instances these diagnoses had not been verified by the Röntgen ray. The average duration of symptoms was twelve years, the shortest duration two months. Several patients had had symptoms for an indefinite period.

Thirty-seven patients gave a history of more or less typical gall-bladder disease; that is, attacks of epigastric colic, and nausea, vomiting, and residual tenderness, belching of gas, and some qualitative food distress. In nine patients the history was so suggestive of ulcer that a clinical diagnosis of ulcer was made, in spite of negative Röntgen-ray findings. In seven cases it was only possible to elicit an indefinite history of an upper abdominal condition which might have been considered as disease of the gall-bladder or stomach, or as reflex in character.

A clinical diagnosis was made of cholecystitis in twenty cases, cholecystitis with stones in fifteen, ulcer in nine, appendicitis in four, and indeterminate in seven. Blood appeared in the vomitus in thirty-two patients, in the stool in eighteen, and in both vomitus and stool in five. It is difficult

to judge at all accurately of the amount of bleeding, as it was only stated that the hemorrhage varied in amount from a few ounces to severe bleeding, producing anæmia.

Operations of drainage and removal of stones from the gall-bladder had been done elsewhere in three instances, and the appendix had been removed in seven instances. In thirteen patients constipation was so marked that they had constant recourse to cathartics.

TABLE I

OPERATIVE FINDINGS IN FIFTY-FIVE CASES

Grade	Cholecystitis	Appendicitis
1	16	17
2	25	11
3	10	11
4	4	1

The discrepancy in the total number of cases of appendicitis is due to the fact that seven patients had had appendectomies previously; moreover, at the time of operation at the Clinic, removal of the appendix was considered unwise or unnecessary in several instances, either because of the condition of the organ or of the patient. Cholecystectomy was performed in fifty-two cases, choledochotomy in four, cholecystostomy in one, and appendectomy in thirty-nine. The pathologic condition in the gall-bladder was reported either chronic or acute.

From the evidence of other observers and from my experience it may be concluded that hemorrhage into the stomach or intestine may occur when the lesion is in the gall-bladder or liver. It is difficult to determine at what point bleeding occurs or whether it is from several points.

Cases of cholecystitis in which there is occasional bleeding into the stomach or intestine should be grouped with the toxic cases of gastro-intestinal bleeding. Undoubtedly the bleeding is due to the effect of toxins from the infected gall-bladder or liver. It is generally known how severe hemorrhages may be in deeply jaundiced patients; sometimes bleeding occurs from all the surfaces of the mucous membrane, and is probably largely because of a changed condition of the blood, due to the presence of bile. It is possible, however, that some other change or disturbance of the functions of the liver may be the factor that results in changes in the blood or other tissue, thus allowing toxic bleeding. As our knowledge of these conditions increases, we look more and more to disorders of the liver to account for the obscure cases. It is possible that an ulcer of the duodenum may have been present in our fifty-five cases and not recognized, but since the bleeding ceased in most instances after operation on the gall-bladder it is fair to assume that the gall-bladder was the important factor.

Obscure gastro-intestinal bleeding occurs with many conditions. Obscure bleeding from œsophageal varices, particularly when associated with the toxic state in cirrhosis of the liver, has been widely discussed. There may be

severe bleeding in cases of anæmia associated with enlargement of the spleen and liver, and in certain cases of renal disease, and hypertension, endocarditis, arteriosclerosis, aneurism, and tabes dorsalis may be causes of obscure gastro-intestinal hemorrhage. In any event, all obscure hemorrhages occurring in the stomach or intestines are not the result of infections in the gall-bladder; they may be the result of disturbances in the liver which have been brought about by any one of these causes.

Moschcowitz reported four cases of massive hemorrhage from the stomach without demonstrable ulcer, in which operation failed to reveal lesions in the stomach or any other condition to account for the bleeding. The patients all recovered after repeated transfusions and Moschcowitz was inclined to believe their condition to be the exulceratio simplex described by Dieulafoy, who, in two cases, had not found a demonstrable ulcer or lesion of any kind in examining the outside of the stomach, but in each instance on opening the stomach and brushing away the clots, a small abrasion of the mucous membrane which extended into the submucosa was found. In one of the cases Dieulafoy was able to show a small open arteriole at the bottom of the small break in the mucosa and submucosa. This probably constitutes a true, although minute and superficial, ulcer, and demonstrates that a great deal of bleeding may occur from a very small opening in the tissue. I believe it should not be assumed that this type of lesion accounts for all the cases of obscure bleeding; if it did, the condition would be reported more often at necropsy.

Apparently there are three regions in which similar obscure bleeding occurs. Bleeding from the stomach or intestine, when the mucous membrane is intact, may be comparable to bleeding from the nose when the mucous membrane of the nose and throat is intact, and is also comparable to bleeding from the kidney which is designated essential hæmaturia. In these three conditions the blood escapes from apparently normal mucous membrane. Although the tissues are swollen and œdematous, they are intact, and there is no way to explain the bleeding, except by oozing. Although the evidence is a little uncertain, I believe the cause is generally considered to be toxæmia, probably originating from any one of a number of sources and producing the same effect.

Many times the wrong diagnosis has been made through the error of thinking that if bleeding were associated with dyspepsia there must be ulcer in the stomach or duodenum. If blood was vomited the diagnosis seemed positive, but we now regard this symptom, and that of blood from the bowels, as occurring in association with other conditions in a certain percentage of cases. An abdominal exploration does not seem justified on the evidence of the bleeding alone, as the source of the difficulty may be more remote. However, since infections in the gall-bladder, biliary tract, and liver are undoubtedly often responsible, I believe that in operating on a patient with these symptoms, the biliary tract should be carefully investigated, whether or not there is an ulcer. It has been shown that twenty per cent. of patients

with duodenal ulcer have noticeable bleeding. Most of these patients are completely relieved of all their symptoms by gastro-enterostomy. In from about ten to fifteen per cent. of such patients bleeding recurs at some time after the operation. In most of these the bleeding may be the result of latent or revived infection in the duodenum at the site of the ulcer. It is possible, however, that it may sometimes be due to infection in the gall-bladder and that it might not have occurred if an associated infection in the gall-bladder had been recognized at the time of the operation for the ulcer. In operating on the stomach or duodenum it has been our custom to examine the gall-bladder and ducts, but I believe it is possible to overlook inflammation in the gall-bladder, unless we recognize the lesser degrees of inflammation that may occur.

We have an abundance of evidence to show that cholecystitis and hepatitis may be the source of the infection which results in bleeding, and I think we also have evidence which suggests that cholecystitis may be the source of the infection, causing this symptom even in the presence of ulcer of the stomach or of the duodenum.

REPORT OF FOUR CASES OF BLEEDING ULCER OF THE DUODENUM ASSOCIATED WITH CHOLECYSTITIS

Case I (A364189).—Mr. F. A. H., aged sixty-one years, came to the Clinic July 9, 1921, complaining of having had hunger pains for the last two or three years which disappeared on eating. He was supposedly cured by a gastro-enterologist, but about a month before our examination he began to lose his appetite, and his old discomfort returned. He noticed that his stools were dark-colored for several days. On two occasions he had had hemorrhage from the stomach, once with momentary loss of consciousness and once for five minutes. He had been on a liquid diet for some time and during the two weeks before examination had lost six pounds.

Examination revealed blood-pressure of 175 systolic and 100 diastolic. The prostate was somewhat enlarged, and the phenolsulphonephthalein return on two occasions was thirty and thirty-five per cent. Hæmoglobin was forty-three per cent. Röntgen-ray examination confirmed the presence of a duodenal ulcer.

At operation July 16, 1921, an ulcer of the duodenum was found on the anterior wall about 1.5 cm. below the pylorus; this was causing obstruction and dilatation of the stomach. The gall-bladder was infected and contained multiple stones. Gastro-enterostomy was performed and the gall-bladder removed. The patient's convalescence was uneventful, and he has been entirely well thus far.

Case II (A325048).—Mr. H. Q., aged fifty-eight years, came to the Clinic July 15, 1920. He had always been well until three years before, when he passed dark stools, and was very weak afterward. Two years later he again passed considerable blood by bowel. He was put on a diet and improved rapidly. In March, 1920, while in Russia, he had another attack of severe bleeding from the bowel. He became very weak and his hæmoglobin dropped to thirty per cent. For about three weeks preceding the second attack he had had epigastric pain. Since his last attack he had had considerable dyspnœa and asthmatic-like attacks.

Examination revealed an anæmic man weighing 208 pounds, somewhat less than his normal weight. The systolic blood-pressure was 108, the diastolic 84. There was a blowing systolic murmur all over the chest and carotids. Urinalysis revealed albumin, casts, and pus. The hæmoglobin was thirty-eight per cent.,

coagulation time five minutes, and bleeding time one-half minute. Röntgenograms showed an ulcer of the duodenum. The electrocardiographic examination showed ventricular preponderance 3, on a scale of 1 to 4, chronic myocarditis, and aortic stenosis. The teeth and tonsils were septic.

At operation, August 6, 1920, a diffuse superficial ulceration on the anterior wall of the duodenum of the duodenitis type was found. The ulcer started about 1.25 cm. below the pylorus and extended for 5 cm., there was very little if any induration. The gall-bladder was definitely inflamed and contained stones; the liver was inflamed. A posterior gastro-enterostomy and a cholecystectomy were performed. The patient's convalescence was normal and he left the hospital in about two weeks. Several weeks later his tonsils were removed and his teeth treated. He recovered completely.

Case III (A355215).—Mr. F. D. McC., aged forty-one years, came to the Clinic April 12, 1921. He complained chiefly of gas in the stomach and epigastric distress which had been intermittent for four years. The attacks came usually during the spring and fall and lasted for from three weeks to three months. At one time he was free from trouble for two years. The pain usually came on about five o'clock in the afternoon and then again from one to three in the morning. Relief was obtained by food and soda. He had his first hemorrhage from the stomach in 1919 and the second in 1921, when he also passed dark stools.

The patient was found in good condition except for his anæmia. The hæmoglobin was forty-two per cent. (about two weeks after his last hemorrhage). Röntgenograms revealed an ulcer of the duodenum.

Operation was performed April 20, 1921. On exposing the duodenum an old œdematous ulcer was found on the anterior wall of the duodenum which was attached under the liver. The gall-bladder was definitely inflamed and contained stones. The appendix was also subacutely inflamed. A posterior gastro-enterostomy was made and the gall-bladder and appendix was removed. The patient recovered completely.

Case IV (A355365).—Mr. F. S. K., aged fifty-nine years, came to the Clinic April 14, 1921, complaining of recurrent hemorrhages from the stomach and bowel. The history of his trouble started thirty-three years before when he first had attacks of epigastric pain. The attacks lasted for from one to six months; he was then free from trouble for from one to two months, during which time he felt perfectly well. Epigastric pain began from two to three hours after eating and often wakened him at night. Food and soda afforded complete relief. He did not vomit during the attacks of pain, but had much belching of gas. These spells persisted intermittently for years. Nine years before he had passed tarry stools for five or six days and became very weak. Shortly after this he was operated on and a duodenal ulcer found. A gastro-enterostomy was done elsewhere. The patient was relieved for eight months, and again had trouble, but the attacks were shorter and less frequent than before the operation. They were relieved by soda and food. After the operation he had hemorrhages about once in six months, as evidenced by tarry stools, weakness, dizziness, and dyspnœa. He was treated medically on a number of occasions and was relieved of his symptoms. While on the train on his way to the Clinic he had passed profuse tarry stools; this was followed by weakness and dizziness. Increasing pain had persisted for many months. He had lost fifteen pounds in weight in the last four months.

At the time of our examination he was anæmic and weighed 147 pounds. The systolic blood-pressure was 180, the diastolic 110. An occasional hyaline cast was found in the urine. His hæmoglobin varied from fifty to sixty per cent. The gastric contents showed total acids 32, free acids 20. The combined phenolsulphonephthalein return was fifty-five per cent. in two hours.

The röntgenograms revealed a deformity of the duodenal bulb and a free gastro-enterostomy. Some time was required in raising his general resistance as much as possible for operation.

At operation May 3, 1921, an ulcer was found about one and one-half inches below the pylorus which had constricted the duodenum until it was only a small cord and would apparently permit little or no fluid to pass. This was ligated with two silk ligatures which were placed one above and one below the centre of the contracting scar. Examination of the old gastro-enterostomy showed it to be in good condition, with a large opening and no evidence of ulcer in the margin. The gall-bladder was definitely inflamed and contained one small stone. The liver was œdematous and hardened, with scars on the surface. The ulcerated area was occluded by silk ligatures and the gall-bladder then removed. Convalescence was very satisfactory for the first four days after the operation, when the patient had a sudden attack of abdominal pain, intermittent in character and not localized. On account of the arteriosclerosis we thought for a time that the pain might be anginal. The pain was completely relieved by a hypodermic of morphin, but returned again in a few hours, and his pulse became more rapid.

Leakage was suspected and the abdominal wound opened. The general abdominal cavity was in good condition, but there was a small pocket beside the duodenum containing duodenal contents which had leaked from an area of perforation through the old scar tissue where the ulcer had been. The pocket was walled off and free drainage established. The patient gradually grew weaker and died on the night of the sixth day.

When the duodenum was first examined in this case it seemed best to excise the scar tissue. At least it was considered absolutely necessary to destroy the ulcerated area to obviate the possibility of a return of hemorrhage. When the duodenum was partly mobilized, we realized that further procedure was not feasible, because the dissection of the scar tissue led into the pancreas, common, and pancreatic ducts. The idea of removing the ulcer was abandoned, and we attempted to place ligatures to control the blood-vessels to the ulcerated area. Possibly this reduced the circulation so that necrosis resulted; at least there was some change in the perforation on the fourth day after a normal convalescence to that time.

Necropsy showed that there was no true ulceration in the duodenum; its lumen was greatly reduced, but it was entirely covered by mucous membrane. While this patient had a rather marked arteriosclerosis and a large ulcerating area in the duodenum, cholecystitis with stones and hepatitis, I am, nevertheless, inclined to believe that if he had recovered after cholecystectomy, he would probably have been relieved of his intestinal hemorrhages.

BIBLIOGRAPHY

[1] Balfour, D. C.: Surgical Treatment in the Bleeding Type of Gastric and Duodenal Ulcer. Jour. Am. Med. Assn., 1919, vol. lxxiii, pp. 571–575.

[2] Crispin, E. L.: Toxic Gastric Hemorrhage. California State Jour. Med., 1917, vol. xv, pp. 308–312.

[3] Deaver, J. B.: Gastric Hemorrhage. Surg., Gynec. and Obst., 1914, vol. xviii, pp. 294–299.

[4] Dieulafoy, G.: Exulceratio simplex; l'intervention chirurgicale dans des hématémèses fondroyantes consécutives à exulcération simple de l'estomac. Bull. de l'Acad. de méd., 1898, 3 s., vol. xxxix, pp. 49–84.

[5] Kelling, G.: Die Beziehungen zwischen Cholelithiasis und Ulcus duodeni. Archiv f. Verdauungskr., 1921, vol. xxiii, pp. 63–68.

[6] Moschcowitz, A. V.: Massive Hemorrhages from the Stomach without Demonstrable Ulcer. Am. Jour. Med. Sc., 1916, vol. clii, pp. 714–727.

TRAUMATIC AND INDUSTRIAL HERNIA

REPORT OF THE SPECIAL COMMITTEE OF THE MEDICAL SECTION OF THE AMERICAN RAILWAY ASSOCIATION

WILLIAM B. COLEY, M.D., New York, (Chairman) Chief Surgeon, New York Central R.R.
SOUTHGATE LEIGH, M.D., Norfolk, Va., Chief Surgeon, Virginia Railway,
JOHN B. WALKER, M.D., New York, Consulting Surgeon, Pennsylvania R.R.
C. W. HOPKINS, M.D., Chicago, Chief Surgeon, Chicago & Northwestern R.R.
J. A. HUTCHISON, M.D., Montreal, Chief Surgeon, Grand Trunk Railway.

The great increase in social legislation in recent years has made the subject of traumatic hernia one of vital importance to every industrial organization.

The first Workmen's Compensation Act was passed in Germany in 1884. Similar laws were soon adopted in Austria and later in Denmark, Norway and England. In 1916, thirty-three states and territories in the United States had enacted some form of Workmen's Compensation Act, and since that time, other states have been rapidly following the lead. Therefore, traumatic or industrial hernia, at first largely a question of theoretical interest, has become one of great practical importance. In spite of this, there has been no definite attempt made to standardize our knowledge of traumatic hernia, particularly as regards its etiology. In the recent past, the question of compensation has too often rested upon the power of the plaintiff's attorney to stir the emotions of the jury, rather than upon a carefully-weighed judgment based upon a knowledge of the facts relating to the origin of traumatic hernia. The time has now come when these cases are being gradually taken out of the hands of emotional juries—the members of which, no matter how fair-minded, are naturally lacking in the technical knowledge of the etiology and pathology of hernia—and being passed upon by experienced physicians. Therefore, it is of greatest importance that all of the facts bearing upon the etiology of hernia should be collected and classified and made readily available.

The term "traumatic hernia" has been used in a very general way to include, first, the small group of cases in which the hernia is due to direct violence; second, an occupational hernia, or perhaps, as better classified by the French, "hernia of effort," which includes all of those cases in which the hernia appears during heavy lifting, slipping, falling, coughing, sneezing, or any cause whatever which increases the intra-abdominal pressure; and third, "hernia of weakness" which is due to abnormal or defective development of the abdominal wall at the various hernial sites.

The first group of cases is so exceedingly rare that it may be disposed of in a few words. In true traumatic hernia due to direct violence the tissues must have been punctured by some more or less sharp object which has forced its way at least through the muscles and fascia, if not quite to the peritoneum. Personally, we have never seen a case of true traumatic hernia. We have known of one treated by one of our colleagues; the muscles about the inguinal canal were torn by the horns of a bull and a hernia developed shortly after.

So this group of cases can be practically ruled out of consideration. The latter group, hernia of weakness, due to congenital weakness of the abdominal muscles or weakness through disease, causing atrophy of the muscles, also very rare, as weakness alone without the presence of a pre-formed congenital sac, rarely results in a hernia no matter how great the intra-abdominal pressure. These are practically all of the direct type.

The very large group of cases which is ordinarily designated as traumatic hernia and which should be more properly called occupational hernia, or better still, hernia of effort, furnishes the basis of nearly all of the medico-legal or compensation cases of hernia. The word "rupture," the old English name for the disease hernia, is responsible for the traumatic theory of the origin of hernia so widely held by the laity as well as by many medical men who have given but little study to the subject. This theory gained a foot-hold before operation for the radical cure came into general use and before the etiology of hernia was generally understood. With the rapidly increasing knowledge of the subject derived from a very large number of operations that have been performed in the last quarter of a century, our ideas of the causes of hernia have gradually changed. At present, it is almost universally recognized that the all-important cause of hernia of all varieties is the presence of a pre-formed sac of peritoneum known as the processus vaginalis. This view was held by two noted surgeons of the eighteenth century. Pellatin and Cloquet; but only in recent years did Russell of Australia, by his patient investigations, force us to conclude that practically all hernias are of congenital origin due to this open pouch of peritoneum which has existed since birth. Unfortunately, courts and juries, and compensation laws here and abroad, have not kept pace with the developments of surgery and it is still not unusual to see large damages awarded in cases of so-called traumatic hernia. Russell maintains that an acquired hernia does not exist and recognized authorities on hernia have come to agree with Russell's conclusions.

Prior to the adoption of the Workmen's Compensation Acts, there were a considerable number of medico-legal decisions in cases of so-called traumatic hernia both in Europe and in America. Many of our compensation boards have simply followed along the lines of decisions handed down by European courts. Sheen (*Practitioner,* London, 1909) who has made a careful study of the subject of traumatic hernia in England, states that "the arbiter in these claims, in the mass of ill-understood technicalities, following the lines of least resistance, has given judgment in favor of the workingman—the post hoc ergo propter hoc view being naturally considered the easiest one."

In Switzerland a person suffering from a hernia and desiring compensation, is entitled to indemnity only on the following conditions: (1) It must appear suddenly; (2) It must be accompanied by pain; (3) It must be of recent origin; (4) There must be proof that the hernia did not exist prior to the accident.

In Germany, in order to establish a claim, the sufferer from hernia must have had an examination within forty-eight hours of the accident; the hernia

must have appeared suddenly, must have been accompanied by pain and must have immediately followed some accident. Proof must be furnished that there was no hernia prior to the accident.

While there are no published records showing the results of the New York State Compensation Board, Sellenings, through the courtesy of a medical officer of the commission, has obtained certain important data. The commission thus far has considered traumatic hernia as extremely rare. The opinion was ventured that it occurred in possibly one of ten thousand cases.* Commenting upon these statistics, Sellenings states:

"1. Traumatic hernia is but a surgical curiosity and assumes no practical importance; 2. Only a small number of the cases have been carefully investigated; 3. A great proportion of the cases seem to be relegated to the convenient classification of 'vocational hernias.' Whatever may be said of the attitude of the New York Commission applies equally well to many other sections of the country."

One of the most recent and on the whole judicial discussions of the subject Traumatic Hernia, or, as the author terms it, "Compensable Hernia", is contained in a book on "Industrial Medicine and Surgery" by Harry E. Mock (Assistant Professor of Industrial Medicine and Surgery at Rush Medical College) published in 1919.

Mock calls attention to the fact that "the decisions of established medicine date back to the precompensation days and were based on the testimony of expert authority made in the courts of England especially, and later in our own courts, to the effect that a traumatic hernia could only occur from a direct violence resulting in a definite tearing or rupture of the abdominal wall.

---

* Since this report was made, the following data have been very kindly furnished to us by Mr. Frank V. Whiting, General Claims Attorney of the New York Central R. R. Co. He writes:

"The New York Compensation Commission has never published any statistics or information in relation to hernia.

"For some time, the Board has rather inclined to the view taken in your Committee's report, that is, that hernia is not generally the result of a trauma, yet, there was an unwritten rule established that if the employer offered a correcting operation, and it was refused, an award would be made for eight weeks' disability and the case closed. This practice has prevailed up until very recently.

"In the case of Al. v. P., 223 N. Y. 97, decided two or three years ago, a hernia was claimed as the result of a strain, and the award was made, but subsequently denied by the New York Court of Appeals, on the ground that there was no cause or relation between the strain and the rupture. This resulted in a strengthening of the prevailing opinion that trauma was a doubtful cause of the hernia.

"However, recently, beginning with the case of J. v. D. C., in the Court of Appeals, decided March 15, 1921, there has been a trend the other way. The J. case specifically decided that the facts indicated that the claimant, while lifting a box of clay weighing over 700 pounds, strained his side and a hernia resulted, and the Court stated that there was no doubt that it was an accidental injury within the meaning of the statute, and rendered a decision accordingly."

These quotations are sufficient to show how completely at sea both Courts and Compensation Boards are at the present time, on the subject of Traumatic Hernia.

All other hernias were claimed to be due to congenital defects, pre-formed sacs, and were similar to all other diseases which might occur coincidental with occupation but not related to it. Such testimony was sustained by practically every court and their views were considered as the decisions of established medicine." He states that, naturally few claims for traumatic hernia were made, although employees in those days, just as frequently as at the present time, blamed their work for the condition.

The greatly increased number of claims for compensation for hernia at present, he regards to be due partly to the new attitude on the part of industry in the direction of recognition of certain moral obligations as well as the realization that any improvement in the condition of employees renders them more useful and more efficient. He states, that among broad-minded employers, the question of whether there was such a thing as traumatic hernia for which they could be held legally responsible, caused little concern.

"They were not governed by the decision of established medicine nor of established law but based their decisions upon a just and good business sense. If they employed a man with a hernia they knew the industry was not responsible for it. If it grew gradually worse without any definite accident or excessive occupational effort it was due to natural causes and again they were not responsible. But, if as a result of accident or severe strain this hernia became strangulated, at once doubt as to responsibility entered the case and the decision was, therefore, rendered in favor of the employees. If they hired a man who showed no sign of rupture at his employment examination, but who later suffered an accident or a severe occupational strain and as a result the hernia appeared, compensation and free surgical care were given because in the man's mind the accident caused the trouble, and because they recognized that to a certain extent the occupation was contributory to the final development of the condition.

"From the standpoint of efficiency it was found that a man with hernia was about 25 per cent. less efficient than the man without one. Therefore, these concerns might refuse to employ men with a rupture but they became more and more liberal regarding the repair of such a condition when it developed in an old employee."

Mock states that, "Such was the attitude of several concerns at the time of the passage of the employees' compensation acts. In fact those very laws were an expression of this new humane influence which had entered industry. The administration of these acts were placed in the hands of industrial commissions whose members were laymen rather than lawyers. Influenced by the generous attitude of certain industries, and guided by this sentiment and a consideration of moral rights, combined with their meagre decision of these various commissions were often at variance to those rendered by the courts in the past.

"Thus employees began to seek compensation for many conditions which heretofore had not been considered compensable, and included among these were hernias which developed during employment."

Mock states, "The question of traumatic hernia, therefore, simmers down to three considerations:

1. A proper definition of what is meant by traumatic hernia;
2. To what extent must an accident or an occupational hazard which only partially contributes to the development of a condition be held responsible for same;
3. In which cases should compensation be paid by the employer."

Mock fully agrees with our own opinion and that of practically all surgeons who have had much experience with hernia, that, hernias as a result of direct violence are very rare. He states that many of the best authorities have enlarged the scope of traumatic hernia so as to include those cases which result from the indirect application of force causing greatly increased intra-abdominal pressure. This adoption of a broader definition, however, Mock believes would mean the inclusion of many additional hernias in the compensable group thus greatly confusing the question. We believe it would be much better to restrict the name of traumatic hernia to the very small group limited to direct violence.

Other types of hernia for which the occupation is more or less responsible, are described by Lotheissen and other writers as "accidental hernia."

Mock has personally observed only five cases of true traumatic hernia due to direct violence at the point where the hernia developed. He cites these five examples as follows:

"(1) Man struck in the right groin by the sharp end of a crowbar; (2) a brakeman was crushed between the bumpers of two cars and a ventral hernia appeared; (3) a man was running through the aisle at fire drill and struck his left inguinal and scrotal region against a truck handle. A large contused area, swelling and hemorrhage into the scrotum immediately followed. Within three days a definite left, direct inguinal hernia appeared; (4) a pregnant woman was kicked in her left lower abdomen by her husband and very shortly a ventral hernia appeared and naturally increased in size as pregnancy developed; (5) a cowboy came to my clinic with two enormous oblique inguinal hernias. He gave a history of some two years previously having had a horse he was riding rear and fall over backward, pinning him beneath the saddle. The pommel of the saddle had crushed into his lower abdomen. Immediately there was bulging in both groins and these continued until they had reached the present size. The man denied any sign of rupture previous to the accident."

In at least the fifth case of Mock's series (enormous double oblique inguinal hernias) it would seem almost certain that there must have been present congenital sacs, or rather, an early stage of hernia on both sides prior to the accident, and the enormous increase in intra-abdominal pressure in this case further developed the pre-existing condition. Mock himself admits that, "It is quite evident that even in these cases of inguinal hernia following direct violence, some doubt will always exist as to the possible presence of

a congenital predisposition for hernia." He very truly affirms that, "Industrial Commissions all over the country are depending on the surgeons in industry to arrive at a just and equitable decision concerning this subject of compensable hernia."

Mock believes that, "The first essential is to make a careful physical examination of all employees and to record those who have real or potential hernias. Whenever a hernia develops in one of these employees who was recorded not to have a hernia, a careful analysis of his case must be made to determine: (1) was it entirely due to pre-existing defect? (2) was it entirely due to some severe direct or indirect violence? (3) was a latent condition already present and only aggravated by the unnatural occupational hazard? (4) was it due entirely to natural causes? (5) or was it due to a combination of all of these, and if so, which was the most responsible?"

Mock admits that, "The great majority of hernias develop slowly, 'the gradual dilatation of a pre-formed sac.' The congenital defect or predisposition is the chief cause for such hernias and the relations of natural occupation or of the natural acts of ordinary life are immaterial in their formation. These correspond to the gradual development of 'flat-foot' a result of faulty shoes, constant standing and walking or other natural causes; or to the development of tuberculosis in employees engaged in occupations which in no wise predispose to this condition."

MacCready, the greatest English authority on hernia, states that an acquired hernia is never due to an accident or single increase of intra-abdominal pressure.

Graser, one of the highest German authorities, states that a hernia complete in all its parts can never arise at the moment of accident or by a single increase in the intra-abdominal tension be it ever so great.

Moschcowitz, of New York, who made a very careful study of hernia in relation to the Workmen's Compensation Act (*Med. Rec.*, April 3, 1915), concludes: "Traumatic hernia is exceedingly rare. It may occur in any part of the abdomen, but usually *not* at the site of the normal hernia openings. Workmen's Compensation Commissions are not and cannot be acquainted with all the facts relating to hernia. This is evidently the sphere of the medical profession; the Workmen's Compensation Commission should be required to place implicit reliance upon the decision of established medicine. In cases of appeal from the decision of the Commission, all the medical part of the testimony should be given by experts of the court's selection, and not of the selection of the claimant or defendant."

A fact particularly emphasized by Doctor Hopkins is, that the great majority of hernias in industrial practice, particularly in railroad work, are found in foreigners, and nearly all in men who have not previously passed a physical examination. One of the reasons why they occur more frequently in foreigners is, we believe, the fact that the class of foreigners engaged in the lower grades of railroad labor are, as a rule, either under-nourished at the time, or went through a period of under-nourishment during childhood,

which tended to lessen the normal development of the abdominal wall. Another reason for the higher percentage of hernias in foreigners, particularly those coming from Russia and Southern Europe, may be found in the practice so widely prevalent among these people, of trying to produce artificial hernia in order to escape army duty. Doctor Gerster, of New York, called attention to this factor many years ago; and recently, at the Hospital for Ruptured and Crippled, Doctor Hogust observed a double direct hernia, regarding which the man stated he had produced it himself. The method of production was: taking a hard, slightly blunted stick, placing it over the inguinal canal and then striking moderate blows from time to time with a mallet until the muscular structures in the neighborhood of the canal are torn or pushed to one side and finally a hernia develops. Here again we must observe that it does not occur as the result of a single blow or single injury. It is only the repeated blows with this more or less sharp instrument that finally produce such a weakness as to cause a direct hernia to follow.

Of all the attempts made by the different State commissions to solve this vexed problem of traumatic or industrial hernia, the industrial commissions of Nevada and California stand out as most in accord with our present knowledge of the causes of hernia. The following is a ruling of the California Industrial Commission:

"The consensus of medical and surgical opinion runs to the effect that hernia is very rarely, in any proper sense, the result of an accidental injury; that the accident is at best no more than the occasion, instead of the cause of the malady; that the origin of the difficulty is congenital and more in the nature of a disease than an injury; that every claim for compensation based upon an alleged rupture is to be viewed with suspicion."

The Nevada Commission rules:

"Medical science teaches now what it has taught for the past twenty years and is now accepted as a medical and scientific truth, corroborated as such by the foremost surgeons and anatomists in the world; that is, that hernia, or so-called rupture, is a disease, ordinarily developing gradually, and is very rarely the result of an accident."

The following rules have been promulgated by the Nevada Commission:

"Rule I. Real traumatic hernia is an injury to the abdominal wall (belly wall) of sufficient severity to puncture or tear asunder said wall and permit the exposure or protrusion of the abdominal viscera or some part thereof. Such injury will be compensated as temporary total disability, and as partial permanent disability, depending upon the injured individual's earning capacity.

"Rule II. All other hernias, whenever occurring or discovered and whatsoever the cause, except as under Rule I, are considered to be diseases, causing incapacitating conditions or permanent partial disability, and the causes of such are considered, as shown by medical facts, to have either existed from birth, to have been years in formation, or both, and are not compensatory, except as provided under Rule III.

"Rule III. All cases coming under Rule II in which it can be conclu-

sively proved, first, that the immediate cause which calls attention to the presence of the hernia, was sudden effort or severe strain or blow, received while in the course of employment; second, that the descent of the hernia occurred immediately following the cause; third, that the cause was accompanied or immediately followed by severe pain in the hernial region; fourth, that the above mentioned facts were of such severity that they were noticed by the claimant and communicated immediately to one or more persons, are considered to be aggravations of previous ailments or diseases, and will be compensated as such for time or loss only, depending on the nature of the proof submitted and the result of the local medical examination."

Our Committee is entirely in accord with Rules I and II of the Nevada Commission. It, however, calls attention to a serious conflict in Rule III of the second proof, which must be given in order to establish a right for certain compensation. Rule II states specifically that by medical facts it is shown that a hernia either exists from birth or is years in formation; whereas, in the second proof of Rule III it speaks of a descent of hernia occurring immediately following a strain or blow. This assumes that hernia may be the result of a single increase of abdominal pressure which the Commission in Rule II stated to be impossible.

Many writers state that a recent hernia is tender and painful on manipulation, and ecchymosis is not infrequently present. This statement is frequently found in text-books and particularly in articles upon Traumatic Hernia. We believe it has no basis in fact. In an experience of 31 years at the Hospital for Ruptured and Crippled, where we have an average of 5000 new cases a year, there has not been a single case of recent hernia which was "tender," painful, and accompanied by ecchymosis in which there had been a history of antecedent injury or accident of any form. We have seen a number of cases that were attributed to an injury, and we are of the opinion that the patients honestly believed that the injury was the cause of the hernia; yet the size of the hernial ring, the thickness of the sac, with adhesions to the surrounding structures, all proved beyond the shadow of a doubt, that the hernia was of long standing, although probably not previously recognized by the patient. A recent case, only observed in October, 1921, is a very good illustration of this point. A man, 25 years of age, employee of the New York Central Railroad Company, with a history of never having had any swelling whatever in the region of the hernial canals, shortly after heavy lifting, noticed a swelling in the right inguinal region. He came to the Emergency Hospital of the N. Y. C. R. R. Co., where the attending surgeon found a well-marked inguinal hernia, the size of a small egg, in the right inguinal region, extending well into the canal and upper scrotum. In the opinion of the surgeon, this was one of the most definite cases in his experience pointing to a causal relationship between the strain and the hernia, and it might have been so regarded, had not the patient consented to an operation. On October 14, 1921, Doctor Coley operated and found a pre-formed sac undoubtedly of congenital origin, extending well into the upper scrotum, 2 1-2 inches long and 2 inches broad, considerably thickened, firmly adherent to the overly-

ing cremaster muscle. The nature of the sac clearly proved it to be of congenital origin and, in all probability, the hernia itself had existed for months or possibly years although the patient may never have recognized it until the time of the unusual strain, when a somewhat larger amount of omentum or bowel was forced into the sac, causing slight pain which first called his attention to the hernia.

Hernia is practically always due, first, to the presence of a pre-formed sac or open pouch of peritoneum which, in the inguinal variety, follows the testis in its descent into the scrotum, which pouch has failed to close in the normal way; and second, to the presence of structural weakness in the neighborhood of the hernial orifices due to poorly developed muscles or fascia. Given these all-important anatomical causes which are in themselves sufficient in many cases to constitute a potential hernia, the actual hernia may develop by reason of a great variety of exciting causes; among these may be mentioned, the daily increase in intra-abdominal pressure incident to the ordinary routine of life, e. g., straining at stool, coughing, sneezing, lifting, etc. The main point that cannot be emphasized too strongly is that the hernia is never the result of a single strain or single increase in intra-abdominal pressure due to any of the causes mentioned; on the other hand, it is the cumulative effect of a great number of strains spread over a considerable period of time. In nearly all cases hernia is of gradual onset, and is rarely accompanied by pain, and most frequently remains unnoticed until it has reached a considerable size or until some accident or strain by slightly increasing the contents of the hernia sac, causes it to be noticed for the first time. Hence, the accident or strain is usually the occasion which first attracts the attention to a hernia long present but hitherto undiscovered. It has been a matter of almost daily observation at the Hospital for Ruptured and Crippled to find a patient applying for a truss or for operation for a hernia on one side, when careful examination discloses the fact that he has a hernia on the other side, almost, if not quite as large as that for which he applied for treatment. The size of the hernia and the character of the sac as determined by operation prove beyond question that this hernia existed for a long period and was quite unrecognized by the patient. Hence it is true, that, in many cases a person who claims that his hernia is due to an accident or an injury may sincerely believe this to be the fact, because he was unaware of the presence of a swelling prior to the accident, although it had really existed for months or years before. In many other cases, however, the contrary is true and claim for indemnity or large damages is made upon a corporation, for a hernia which the claimant well knew had existed for a long period prior to the accident. In some cases, evidence of his having worn a truss for a long period of time is apparent. We, personally, have seen many cases of this type in our medico-legal work and in some instances the sympathetic jury has awarded very large damages. In all of our experience we have never seen a single case in which there was any sound basis for the claim that the accident or injury was the cause of the hernia. In many cases the jury has been convinced by expert testimony that a hernia could not have been caused by the accident mentioned

and have rendered a verdict accordingly, but on the other hand, in other cases, all of the expert evidence has been brushed aside and the jury has allowed its sympathy for the claimant to outweigh the seemingly slight loss of a few thousand dollars compensation to the supposedly wealthy corporation. One case which we recall is that of a man of about 50 years of age, who claimed to have been thrown forward against the back of the seat in front of him in a slight collision. The slight increase in intra-abdominal pressure was made the basis for his claim that a large double, inguinal hernia was the result of the accident, although there was no evidence of local injury at the site of either hernia. In spite of expert evidence to prove the fact that a double hernia is never the result of trauma, that these hernias were both too large to have been of recent origin, the jury, as we remember, awarded very large damages ($15,000.00). The verdict was so palpably against the evidence that the decision was reversed by the Supreme Court.

At present the situation in regard to dealing with the question of traumatic or industrial hernia may be described as chaotic. There are, however, a few States in which the members of the Workmen's Compensation Commission apparently have made a scientific study of the subject before formulating any rules and in these States the subject is treated in a most fair-minded and judicial way; in other States, however, the rulings are apparently based on the old and long-discarded ideas as to the etiology of hernia with the result of great financial loss to the interested corporations and in the end distinct harm to the individuals.

What then is the remedy? The only thing needed to bring about greater harmony in the procedure of industrial commissions, is to spread broadcast a clearer knowledge of the well-known medical and surgical facts relating to the etiology of hernia. We must recognize that medical and surgical truths permeate but slowly, especially when they have to overcome long-established traditions too often supported by court decisions. The first is to convince the commissions and the courts of the well-established surgical fact that hernia is a disease and not the result of an accident. When this has been done a radical review of the present State laws regarding compensation in cases of industrial hernia will be forthcoming.

RECOMMENDATIONS

1. Render proper compensation for all cases of true traumatic hernia due to direct violence. Such cases are so few in number as to be practically negligible.

2. Make a physical examination of all applicants for positions in industry no matter in what capacity; such examinations will determine the fact whether or not a hernia was present at the time of examination.

Doctor Hopkins, in a recent paper on Traumatic Hernia, reveals the important fact that of all the men who passed the physical examination prior to entering the railroad service, the claim of traumatic hernia amounted to less than one per cent.; while, on the other hand, ninety-one per cent. of the cases of alleged traumatic hernia were found to occur in foreigners (Greeks,

Italians and Poles), who did not have a preliminary physical examination before being admitted to the service.

3. Any case of hernia developing in the course of duty, incident to the man's daily work, should be treated as a disease due to special anatomical weakness on the part of the individual, for which the Company is in no way responsible. If it is considered wise under certain circumstances to recognize any moral responsibility, let it be on an economic or humane basis. This moral obligation should be understood to be strictly limited to such employees who had been found apparently free from hernia at the time of previous physical examination.

BIBLIOGRAPHY

Berger: Rev. de Chirurgie, 1906, Nos. 4 and 5.
Kocher: Correspondenzblatt f. Schwitz. Aerzte, 26, 1898.
Von Hassel et Walravens: Jour. de Chirurgie, Bruxelles, 126, 1903.
Forgue et Jeanbrau: Accidents du Travail, Paris, 1905.
Berger: Traite de Chirurgie (Masson et Cie.) Dupley et Recluse, vol. vi.
Kingdon: Med. Chir. Transactions, 1864, pp. 286–296.
Murray: Lancet, April 20, 1907.
Roberts: N. Y. Med. Jour., 1904, vol. lxxx, p. 631.
Hamilton Russell: Lancet, 1904, p. 797. *Ibid.*, 1907.
Blasius: Verhandl. d. Gesellsch. Deut. Naturf. u. Aertz., 1895, lxvi.
Stuki: Correspbl. f. Schweitz. Aerta., 1899, p. 589.
Sultan: Abdominal Hernia (Saunders), Coley and Satterwhite, Internat'l Jour. of Surgery, Feb., 1904.
Gallaudet: Med. and Surg. Reports of Bellevue Hosp., 1904, vol. i.
Hernie Considérée Comme Accident des Travail: Jour. de Méd. de Paris, 1907, 2, S. xix. 53.
Lucas-Championniére: Jour. de Méd. et de Chir., prat., 1906, lxxvii, 6.
Reclus: Clinique Paris, 1907, ll 249.
Butte: N. Y. Med. Jour., October 19, 1907.
Cutten, Witthaus and Becker's Jurisprudence, Forensic Medicine and Toxicology, 1907, p. 853.
Daget: Le Hernie est elle accidente du travail. Thèses de Paris, 1905.
Graser: Hand-book of Practical Surgery, 1900, vol. xx, p. 826.
Hopkins: International Journal of Surgery, January, 1921.
Lotheissen: Arch. f. Orthop. Mechanotherap. u. Unfallchirurg, 1906, bd. lv.
MacCready: A Treatise on Ruptures (Blakiston).
Mock: Industrial Medicine and Surgery (Saunders).
Moschcowitz: Medical Record, April 3, 1915.
Sellenings: N. Y. Medical Journal, April 24, 1920, p. 713.
Sheen: Practitioner, London, 1909.
Duchamp: La hernia au point de vue mé légal, Loire médic, St. Etienne, 1900, p. 258.
Gazette médicale de Paris, 1901, p. 170.
Jacquet: Echo méd. du Nord., 1900, p. 500.
Janin: Théses Paris, 1902.
Loriot: Théses Paris, 1902.
De Quervain: De la hernie de force, Semaine médicale, 1900, p. 87.
Socin: Chr. Bl. f. schweiz. Aerzte. Bezel, 1887, p. 545.
Sole: J. de Méd. de Paris, 1904, p. 27.
Coley: International Journal of Surgery, February, 1906.
Coley: International Journal of Surgery, February, 1904.
Coley: Keen's Surgery, vol. iv.

NON-TUBERCULAR KIDNEY INFECTIONS*

By Raymond P. Sullivan, M.D.
of New York, N. Y.

Infection of the kidney by pathogenic microörganisms may take place by way of the urinary passages, the lymphatics, or the general circulation. The hæmatogenous route is especially important on account of its often obscure starting point and its production of a great variety of local lesions. The infectious agents in the blood stream reach the kidney through the renal capillaries, in the form of a single bacterial species in pure culture, or as a mixed infection. Albarran's statement still holds good, that with the exception of lesions produced by the tubercle bacillus, our knowledge of other renal infections is as yet incomplete. The three observations of non-tuberculous kidney infections, reported in the following, concern a case of kidney carbuncle, a fulminating acute and a chronic dead kidney. All three cases were the result of hæmatogenous infection of the organ with pus-producing bacteria.

A great significance must be attached to hæmatogenous infection of the kidney, which is the eliminating organ for microbes in the general circulation and is therefore liable to damage through the bacteria themselves or their toxic products. The starting point of kidney infections is extremely variable and often situated at a considerable distance from the organ. The cortical lesion of the kidney or localization of the blood infection, from which the perirenal suppuration originates, may be so small as to escape detection even at the time of operation. These metastatic infections will give rise either to diffuse suppurative nephritis, to focal suppuration within the renal cortex, or to perirenal or pararenal suppuration with a trifling cortical lesion. All these conditions are merely different degrees of one identical process, namely the localization in the renal cortex of pathogenic agents which have been carried there by the blood stream in the course of bacteriæmia, usually staphylococcus bacteriæmia. Metastatic hæmatogenous infection of the kidney or perinephritic abscess secondary to a small pus focus elsewhere in the body is not always easily recognized, but on the contrary in acute cases lends itself readily to confusion with acute intra-abdominal infection.

The name of metastatic carbuncle of the kidney was first applied by Israel to metastatic hæmatogenous abscess of the renal parenchyma and perinephritic abscess, which may follow upon practically all suppurative or infectious processes of the rest of the body, especially boils and felons. An instructive example of perinephritic abscess which was later found to have an associated carbuncle of the kidney, recently came under my own observation, offers a good illustration of this not uncommon hæmatogenous origin of kidney suppurations.

* Read before the Southern Surgical Society, December 13, 1921.

CASE I.—The patient, a young man of nineteen years, sustained a punctured wound of the left forearm, which became infected and was incised by his physician. Some days later he began to complain of severe pain in the mid-abdominal region, and was referred to me at St. Vincent's Hospital. This pain finally localized under the left costal margin, with occasional sharp twinges radiating down the left thigh. On the third day of the abdominal pain, the patient had a chill, and his temperature rose to 103° F. Murphy's sign was present in the left costo-vertebral angle. Urinary symptoms were altogether absent. The diagnosis was perinephritic abscess and the treatment consisted of incision and drainage. The staphylococcus aureus, which had previously been recovered from the infected wound of the forearm, was also recovered from the kidney pus. His post-operative course was quite irregular. Temperature after operation dropped to 101° F., but on the third day it rose again to 103° F. Drainage was satisfactory for eight days, but his irregular temperature and negative digital exploration of sinus for pus pockets, together with negative urinary findings, suggested further urologic investigation. Cystoscopic examination by Dr. Herbert Mohan, on the thirteenth day post-operative revealed equal function from both kidneys. The urine still free from pus and bacteria. However, a pyelogram of the left kidney showed a small renal pelvis, with upper and lower calyces long and narrow, but no sign of middle calyx. The following day left nephrectomy for carbuncle of the kidney was done, and the patient made an uneventful recovery. His present condition shows absence of all symptoms and a gain of forty pounds in weight. The value of urologic examination after drainage of a perinephritic abscess, where the patient presents signs of a continued sepsis, cannot be overemphasized.

The specimen of this solitary staphylococcus suppuration in the kidney (Fig. 1), derived from a remote pus focus, is very striking. The organ is the seat of a peculiar lesion which in color and consistence contrasts so sharply from the surrounding healthy tissue as to suggest a new growth rather than a suppurative process. Beginning in the renal cortex near the surface, the process has developed essentially at the expense of the cortical tissue, advancing inwards towards the central area of the kidney.

Similar conditions existed in Israel's classical case of metastatic carbuncle of the left kidney, observed in 1891 and reported ten years later in his Klinik der Nierenkrankheiten (1901). He emphasizes that this observation concerns the first case on record in which this process of metastatic infection by the blood stream could be demonstrated and points out that undoubtedly hæmatogenous infection may be restricted not only to a single kidney but even to a circumscribed portion of the organ.

A different and more advanced degree of metastatic hæmatogenous infection of the kidney is represented by a personal observation in a case of acute fulminating kidney in a man 57 years.

CASE II.—The patient twenty-four hours before admission to St. Vincent's Hospital was suddenly attacked by sharp pain in the lower right lumbar region, radiating into the right groin and the testicle. These painful attacks lasted half an hour to an hour and were repeated several times daily, becoming progressively more severe, and associated with vomiting, nausea, and hæmaturia. He gave a history of an accident several years previously in which his right hip and a rib were fractured, and remembered a sudden attack of sharp pain in the lower right quadrant in the course of convalescence.

Cystoscopic examination by Doctor Mohan revealed considerable pus coming down from the right kidney. The function of the right side considerably lower than the left. Right pyelogram showed a marked dilatation of the pelvis and ureter with indefinite obstruction in the lower third of the ureter about one and one-half inches from the bladder; one suspicious small scratch on right ureteral catheter. The general condition of the patient became more serious every hour, and operation advised. The next day nephrectomy was done. The kidney found to be considerably enlarged with several areas of subcapsular hemorrhage. At the center there is a large soft area, bluish-black in color, about the size of a walnut. The pelvis is greatly distended. No evidence of calculus or stricture found. The ureter likewise considerably distended and an associated periureteritis extended from the lower third up to and involving the pelvis. Pathological examination of the kidney showed marked changes of acute pyelonephritis with an exudate of pus in the ureter and pelvis extending into the parenchyma, involving the tubules and interstitial tissues, and extending up to the renal cortex. The patient's recovery is especially gratifying in view of the fact, as emphasized by W. J. Mayo, that in the fulminating type of hæmatogenous pyogenic infection, unless nephrectomy is done, death may result within a few days. Observations like the foregoing are more or less suggestive of latent microbism, a problem which attracted much attention during the World War in connection with the healing of wounds. In Brewer's case of acute hæmatogenous infection of one kidney in a woman of twenty years, the condition was preceded about three weeks by double femoral fracture and multiple contusions of the body.

CASE III.—The following case of chronic infectious nephritis and pyelo-nephritis, culminating in "dead kidney," occurred in a man of seventy years whose distressing condition was greatly relieved by drainage of the pus. This patient had suffered five years from urinary retention and when first seen had a persistent sinus of the right kidney of two years standing with a suprapubic fistula of the same duration. Three years previously he was taken suddenly ill with pain and tenderness in right hypochondrium and lumbo-dorsal region. This later proved to be a perinephritic abscess, which was opened and drained through an anterior incision. At the same time the operating surgeon performed a suprapubic cystotomy, evidently because of his attacks of urinary retention. His convalescence was protracted because of remittent fever and backache. The kidney sinus finally healed but opened again one year later just after a prostatectomy by still another surgeon.

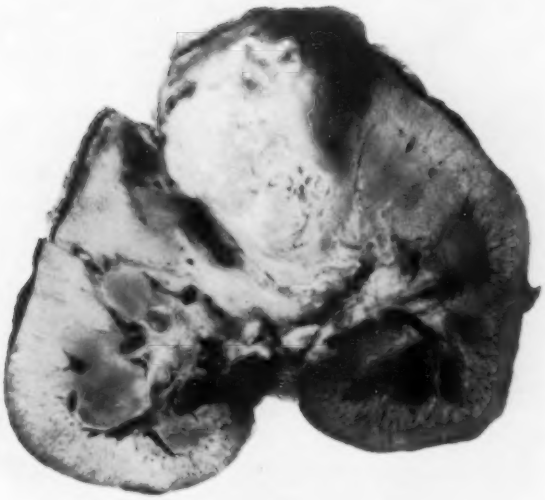

FIG. 1.—Kidney carbuncle. (Cross section.)

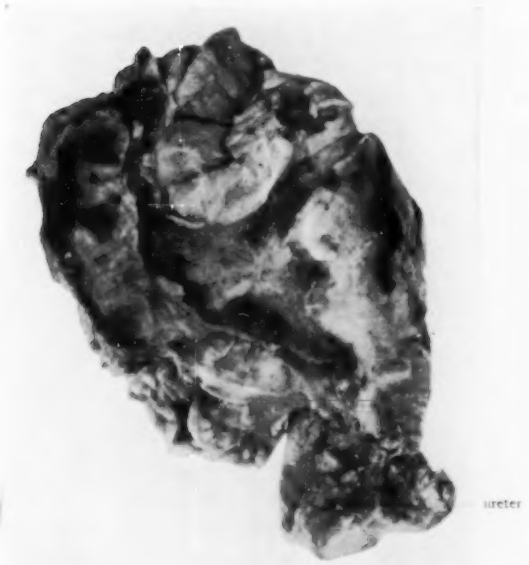

FIG. 2.—Terminal pyelo-nephritis, dead kidney.
(Cross section.)

When first seen by me the patient was bedridden, presenting two discharging sinuses, one from the kidney incision and the other from the suprapubic. There was a constant discharge of pus and urine from a urethral catheter which was kept in the bladder. Cystogram revealed his bladder capacity of about one drachm. He was considerably benefited by our first treatment, which consisted of enlarging the kidney sinus and removing several sloughs. About six weeks later right nephrectomy was performed and a dead kidney removed. His convalescence was quite satisfactory except for a persistent fistula which was found to lead to the ureter stump. Seven weeks after the nephrectomy our secondary operation was performed to remove the ureteral stump containing a calculus. Notwithstanding his advanced years and prolonged sufferings, the patient finally made a good recovery, gained in weight, and was able to go about with all fistulæ closed. His bladder capacity on discharge was sixteen ounces. The findings on macroscopical and microscopical examination were as follows:

Macroscopic Examination.—The specimen consists of right kidney. With the surrounding fat it measured 12 x 9 x 4 cm. The kidney proper measured 9 x 3 x 2.5 cm. It is very irregular in shape and nodular. The parenchyma is almost completely replaced by fibrous tissue, containing several small cysts near the surface. The pelvis and upper part of the ureter are greatly dilated and surrounded by adipose tissue. The mucous membrane of the pelvis is rough and covered with grumous exudate.

Microscopic Examination.—The sections show most of the glomeruli to be completely obliterated and replaced by hyaline connective tissue; some show beginning fibrosis with a thickened Bowman's capsule; none appear to be normal. The tubules show the various stages of degeneration from a granular change in the epithelium to complete destruction. Some of the tubules are cystic and contain an exudate consisting of pus cells and granular material. The interstitial tissue shows generalized lymphocytic infiltration. Many areas show localized infiltrations consisting of lymphocytes, polymorphonuclear neutrophiles, plasma cells and eosinophiles. There are several areas of extravasated blood of recent formation. The mucous membrane of the pelvis is replaced by a membrane consisting of plasma cells, eosinophiles and lymphocytes. There is considerable perivascular infiltration throughout the organ.

Although serious lesions like those described above calling for the removal of the infected organ are a frequent sequel of the penetration of pus-producing bacteria, it is entirely possible for the ordinary pyogenic micro-organisms, especially the staphylococcus, to pass through the kidney without necessarily giving rise to important anatomical lesions.

The *pathogenesis* of deep abscess in general and perinephritic suppuration in particular, was not well understood prior to Pasteur's discovery of the staphylococcus in the pus of the furuncles and its demonstration as the responsible agent in these and other suppurative processes (1881). Some years later, Verneuil in France pointed out on the basis of an illustrative observation that the germs of furunculosis may find a favorable culture medium in a

weakened organism and may multiply freely at a considerable distance from their primary focus, giving rise to a collection of pus. His patient was a physician thirty-two years of age who suffered from a voluminous perinephritic abscess, as a sequel of a furuncle on the upper lip, three styes of the eyelids, and a boil on the cheek. Albarran one year later (1889) applied experimental injections of staphylococcus and colon bacillus cultures into the ear vein of rabbits, and by inflicting lumbar contusions in these animals succeeded in producing perirenal suppurations from which the injected pathogenic agent could be recovered. This observation established the fact that perinephritic abscesses can and do follow not only upon an infected wound of the perirenal region, but also upon a blood infection derived from an organ situated at a distance from the kidney. The source of the infection, may, accordingly, consist not only of a severe general disease but a distant and perhaps apparently insignificant focus is often responsible. In such cases perirenal localization of the germs is favored by more or less definite factors, such as traumatism or exposure.

Etiology.—A distinction must be made between abscesses propagated to the kidney from other organs by continuity and abscesses of purely metastatic character which form in the renal cortex through embolic transportation of infectious agents in the renal arteries. These abscesses are usually due to the staphylococcus aureus, as in my own observation, and often assume a relatively mild type, whereas acute streptococcal infections of the kidney, as pointed out by W. J. Mayo are apt to be extremely malignant and are fortunately rare. Pus-producnig bacteria, which reach the kidney parenchyma by the hæmatogenous route, are apt to lead to the formation of metastatic parenchymatous, perinephritic, or paranephritic abscesses; and these may follow upon practically all suppurative or infectious processes in the entire body. Traumatism in the form of a blow or kick in the renal region sometimes acts as a determining factor in the localization of the lesions.

It is now understood that the pathogenic micro-organisms which have penetrated into the blood current may pass through the kidney, which is the eliminating organ for microbes in the blood current, without giving rise to serious lesions of the kidneys themselves or the excretory passages, and without apparently modifying the composition of the urine. Existing lesions of the renal cortex may be so trifling as to escape detection on superficial examination. The character of the pus in these infections naturally varies according to the character of the blood infection. The staphylococcus aureus is usually found following upon boils and felons, while in other cases the streptococcus, the pneumococcus, the gonococcus, the colon bacillus or anaërobes may be demonstrable. The two latter are found more particularly in perinephritic suppurations following upon lesions of neighboring organs, such as the colon or duodenum.

The subject of metastatic hæmatogenous perinephritic abscess has recently been discussed by Cleisz, in a Paris Thesis (1919), who reports six unpublished cases of staphylococcus aureus infection from the service of Lecéne.

One of these cases was observed and operated upon before the war in 1913; the five others came under observation and treatment during the war. Four patients gave a history of felons or boils, one had previously suffered from an attack of influenza, and one from severe tonsillar angina with metastatic suppuration of the elbow. It is important to keep in mind that metastatic perinephritis may be a sequel of practically all suppurative or infectious lesions of the entire body, notably boils and felons, but also general diseases such as typhoid fever, influenza, measles, smallpox, gonorrhœa, peritonitis, empyema. The most important and perhaps the most frequent mode of origin of kidney suppuration, accordingly, is hæmatogenous infection in the form of metastatic parenchymatous or perinephritic abscess.

Symptoms and Diagnosis.—The clinical picture of the disease was well described several years ago by Albarran, and no essential addition has since been offered in the literature. The onset is insidious, and the behavior of the temperature is irregular. Pain, although the earliest and most constant symptom, often the only one during several days or weeks, is likewise very variable and may be deep, dull and diffuse, or sharp and radiating in different directions according to the seat of the abscess. Deep pressure in the costo-lumbar angle usually elicits or aggravates the pain. Brewer has pointed out that a marked costo-vertebral tenderness is the one pathognomonic sign present in all cases. In advanced cases percussion with the patient in a suitable position often yields absolute indefinitely outlined dullness. In the extra-renal infections, lumbar swelling is very important and when fairly well marked, may obliterate the costo-iliac notch or even produce a visible lumbar protuberance. Œdema of the skin is very important and constitutes a valuable sign, which alone permits the differentiation of perinephritic abscess from intra-abdominal infection. Cutaneous reddening and fluctuation which may make their appearance in abscesses, with a tendency to point posteriorly towards the skin of the lumbar region, are late symptoms and should never be given time to develop.

A sudden attack of pain in the kidney region, especially when associated with fever, in an individual known to have a suppurative process somewhere in the body should at once give rise to a suspicion of metastatic abscess in or about the kidney. The seat of the pain varies with the location of the focus and may be lumbar or abdominal. The patient's complaints are often misleading, so that the condition may be confused with gall-stone disease or other abdominal affections, which moreover sometimes complicate the renal infection. The diseased kidney is not always indicated by the location of the pain, which has been known to occur on the healthy side, but the modified function of the affected organ will be indicated by the renal functional test, even if the cortical lesion is slight (Braasch).

Changes of the Urine are usually inconsiderable and may be altogether absent. Serious alterations, pointing to an involvement of the kidney, have been noted only in exceptional cases. Microscopical hæmaturia, a few leucocytes, or traces of albumin, are occasionally found, and are sometimes

temporarily demonstrable in the urine. When the urine is normal and there is nothing in the patient's history to draw attention to the kidney, the diagnosis may prove very difficult, although much valuable information can often be obtained through cystoscopic and bacteriological examinations, renal functional tests and radiography.

Differential Diagnosis.—Urological evidence must be carefully secured, for it is only by the functional examination of the kidneys that the cause of the persistent fever can be traced to a destructive process in the kidney. A complete examination includes catheterization of the ureters, a report of the findings in the two separate urines, cystoscopy and uretero-pyelography. In the carbuncle case described above cystoscopy and pyelogram proved the conclusive factor of assistance for the necessity of further surgical intervention. Pyelography is helpful in ascertaining the degree of renal destruction. In advanced cases, the dilatation of the pelvis and calyces may be so considerable as to assume the proportions of a pyonephrosis.

Cystoscopic and other urological methods of examination may be unsuccessful for the reason that the focus is closed off from the outside and does not communicate with the efferent urinary passages. Gross changes of the urine, indicating renal involvement, are only exceptionally present.

In obscure cases, exploratory exposure of the suspected kidney may be necessary. Incision of the abscess alone, without exposure and inspection of the organ, cannot be relied upon to reveal the actual condition of the infected kidney. Fever, sometimes chills, and slight tenderness on pressure may be the only symptoms until the peritoneum becomes involved, when another difficulty arises in the differentiation of the kidney lesion from the diseases of other organs in which the peritoneum is apt to participate. On the right side, which according to some observers is much more frequently affected than the left side, cholecystitis and appendicitis must be excluded. It is also necessary to exclude typhoid fever, malaria, general infections of undetermined character, as well as all conditions associated with backache of a diffuse bilateral type. The backache of perinephritic abscess is localized in the costo-lumbar angle. The formation of a swelling in the renal region may suggest some intrathoracic, pleural or pulmonary affection or an abscess of the lumbar wall. Erroneous diagnosis of disease of the abdominal organs, notably the liver, gall-bladder, or appendix, must be carefully avoided. However, as pointed out by Braasch coincident infection in the gall-bladder or appendix as well as the kidney is not infrequent. Tenderness on pressure below the twelfth rib, and sometimes considerable increase in the size of the kidney, are valuable signs pointing to the existence of an intrarenal abscess.

The differential diagnosis loses some of its difficulties when the possibility of perinephritic abscess is kept in mind, especially when the patient's previous history reveals the existence of a small suppurative process, notably a boil or felon. A sudden onset of lumbar pain in these cases is supposed to correspond to the development of the cortical abscess.

The rendering of an early correct diagnosis is of the utmost importance,

for surgical intervention is indicated. As stated by Keyes, perinephritic abscess should be diagnosed and drained as early as possible. Spontaneous absorption comes too late and is too uncertain.

Treatment.—The treatment of these infected kidneys, which have gone on to localized or generalized pus formation may be obtained by nephrotomy, resection of the kidney or nephrectomy. I agree with Keyes that it will frequently be wise to postpone nephrectomy until drainage shall have relieved the patient of his acute sepsis.

Resection has sometimes been followed by recovery, but the toxæmia is apt to persist for several weeks after the operation, because septic foci have been left behind, so that extirpation of the entire diseased kidney is often preferable especially in weakened patients. Nephrotomy, or resection exposes to the disadvantages of slow healing and secondary hemorrhage; moreover, it is not free from the possibility of a persistent fistula. It enters into consideration, however, as a conservative procedure in those cases where the infectious process is not widely distributed. The removal of the diseased organ prevents the distribution of the suppurative process in the body and protects against the involvement of the opposite kidney. In a general way, and on the basis of numerous considerations nephrectomy is the simplest, most radical, and most promising procedure.

CONCLUSIONS

1. That the kidney is the eliminating organ for circulating microbes, and in the course of this elimination may itself be damaged in a variety of ways.

2. Hæmatogenous infection may be restricted not only to a single kidney, but even to a circumscribed portion of the organ.

3. The source of the infection may not only be a general disease, but a distant and apparently insignificant focus may be responsible.

4. Metastatic hæmatogenous infection of the kidney perinephritic or paranephritic abscess is not always easily recognized, and may be confused with intra-abdominal infections.

5. A sudden attack of pain in kidney region associated with fever in a patient known to have a suppurative process elsewhere in the body should excite suspicion of metastatic kidney infection.

6. Cystoscopy and pyelography are valuable aids especially when urinary changes are incomplete, or the symptoms are referred to the healthy side.

7. The treatment of perinephritic or paranephritic abscess is early drainage. Where the suppuration involves the kidney parenchyma, or where the process is an acute fulminating one—nephrectomy is indicated.

BIBLIOGRAPHY

Albarran: Infectious renales. Traite de Chirurgie, Le Dentu Delbet, vol. viii, 1899, p. 731.

Barth: Der Nirenkarbunkel. Archiv fur klin. Chirurgie, 1920, vol. cxiv, p. 77.

Bilhaut: Abscess perinephritique consecutif a une plaie infectie de la jambe gauche. Annals de Chir et D'Orghoped, 1910, vol. xxiii, p. 2.

Braasch, W. F.: Perinephritic Abscess. Surg., Gyn. and Obst., 1915, vol. xxi, p. 631.

Braasch, W. F.: Infections of the Renal Pelvis and Ureter. Texas State Journ. of Medicine, 1913–14, vol. ix, p. 305.

Brewer, G. E.: Acute Unilateral Hæmatogenous Infection of the Kidney. Surg., Gyn. and Obst., 1908, vol. vi, p. 699.

Cléisz, L.: Les perinephritis suppurees metastatiques. Thèse de Paris, 1919.

Israel, J.: Hæmatogene Nivrenabszesse. Chiruvgische Klinik der Nicrenkrankhiten, 1901, p. 34.

Keyes, E. L.: Perinephritis. Urology, 1917, p. 363.

Mayo, W. J.: Hæmatogenous Infection of the Kidney. Journ. Am. Med. Assoc., 1919, vol. dccxxxii, p. 1023.

Pedroli: Richerche su alcuni casi di infezione renale ematogena. La Clinica Chirurgica, 1920, vol. xxxvii, p. 388.

Verneuil: Des Absces profonds et lointains consecutifs a' l' anthrax. Comptes Rendus Acad. des Sciences, 1888, vol. cvi, p. 112.

NOTE ON THE DIAGNOSIS OF SHADOWLESS RENAL CALCULI*

WITH ESPECIAL REFERENCE TO THOSE OF CYSTIN COMPOSITION

By Roger Colgate Graves, M.D.

of Boston, Mass.

It is common knowledge that a small percentage of renal calculi fail to cast shadows in an X-ray plate. Holmes and Ruggles state that with satisfactory technic "probably eighty to ninety per cent. of kidney and ureteral stones will show." Those occasional calculi which present no greater density than the body soft parts are chiefly of uric acid or urate composition. Much has been written concerning the diagnosis of these elusive stones. The two cases herewith presented, however, offer an interesting point in diagnostic technic which has not been frequently emphasized.

Case I.—(Surgical No. 14733.) A Russian housewife, aged twenty-six, entered the hospital complaining of pain in the right side. Her family history was unimportant. She had had measles, mumps, and scarlet fever in childhood, and tonsillitis on several occasions in later years. At the age of twenty-three she had influenza with pneumonia, and eighteen months before admission a thyroidectomy was performed.

Two years before coming to the hospital she first complained of slight pain in the right upper abdomen and in the lumbar region upon the same side. This discomfort was of frequent occurrence, and it was often associated with activity or fatigue. It was never present at night, and could always be relieved by lying down. The attacks were very transient, and the patient, who was pregnant, attached to them little significance. The pains were occasionally accompanied by vomiting. They bore no apparent relationship, however, to activities of the gastro-intestinal system. There was no jaundice. Save for slightly increased frequency of voiding, ascribed to pregnancy, there were no urinary symptoms.

One year before admission there occurred suddenly at night an attack of very severe pain in the right upper abdomen beneath the ribs, and radiating posteriorly to the back. The suffering was of several hours' duration, and relief was obtained only through morphia. The patient vomited repeatedly; she felt alternately cold and warm. She complained of great urgency associated with inability to void more than a few drops at a time. She did not observe the urine carefully but no blood was seen. The attending physician told her that she probably had a kidney stone. The pain gradually disappeared. The slight twinges previously described, however, continued without change, and after several months there occurred a second period of intense discomfort, comparable to the first one, but less severe.

For several weeks before hospital entry, the pains were of more frequent occurrence, and the patient finally applied for treatment at the Out-door Clinic. There, X-ray studies were negative, although there was slight movement during the examination and the plates were not completely satisfactory. Because microscopic blood was found in the urine, admission to the wards was advised.

Examination found an obese young woman, whose appearance was not suggestive of distress or critical illness. Temperature, 99°; pulse, 92; respirations. 20. The neck bore the scar of a thyroidectomy. The abdomen presented

* From the Urological Clinic of the Peter Bent Brigham Hospital.

slight tenderness and resistance to palpation, in the right upper quadrant. The kidneys were not felt, although obesity made accurate observations difficult. There were no other significant physical findings. The urine was alkaline, clear and pale, and contained neither albumen nor sugar; specific gravity, 1.012. A few white blood-cells were found in the sediment. The excretion of phenol-sulphonephthalein in two hours was thirty-five per cent.; hæmoglobin, ninety per cent.; white blood count, 7600.

Cystoscopy found the bladder normal. Both ureters were readily catheterized. Microscopic examination found the urine from the right kidney to contain many red blood-corpuscles, and no pus. An occasional red cell was found in the specimens from the bladder and left kidney. In the test of divided function the dye appeared promptly and with excellent intensity upon the left side. The excretion in ten minutes was five per cent. Upon the right side fifteen minutes elapsed before any color could be seen and then but a faint trace appeared. All cultures were sterile. Satisfactory X-ray studies confirmed the negative observations previously made in the Out-patient Department. X-ray report: "Plates with the catheters *in situ* show distinctly the outlines of the right kidney. The kidney shadows are not enlarged and there is no evidence of a calculus shadow within either kidney region, along the course of the ureters, or in the bladder."

A pyelogram was made upon the right side. At first glance the result was normal in appearance. There was not the slightest evidence of dilatation of the renal pelvis; the calyces were slender and cupped. In the centre of the main collecting portion of the pelvis, however, where the depth of fluid should be greatest and the shadow therefore densest, closer scrutiny found an oval area of definitely decreased density. If a large air bubble had been introduced at the time of injection it might have been given a similar picture (Fig. 1).

Following this examination, the patient complained of recurrent slight attacks of her typical pain. Several days later cystoscopy was repeated. A wax-tipped catheter was passed along the right ureter and a second right pyelogram was made. The plate was an exact duplicate of the first one, above described. The wax bulb showed linear scratches which were unmistakably of calculus origin.

The two pyelograms were thought to denote the presence of a shadowless calculus within the pelvis of the right kidney, appearing as it were by negativity, through displacement of the denser opaque solution. Holmes and Ruggles state that "papillomata may produce round holes in the thorium shadow." In this instance, however, the patient's story and the wax-tip scratches were sufficient evidence for the exclusion of tumor and the diagnosis of stone. A right pyelotomy was therefore performed and a calculus was removed from the pelvis of the kidney. The operation was without noteworthy incident. The convalescence was equally uneventful, and the patient was discharged to her home on the eighteenth post-operative day.

The stone measured 1.7 cm. x 1.7 cm. x 1 cm. It weighed 2.16 grams. It was lemon yellow in color and glistening as though dusted with sugar (Plate I). In composition it was pure cystin. (Analysis by Dr. Cyrus H. Fiske, Department of Biological Chemistry, Harvard Medical School.)

Cystin calculi are rare, and they have not been thoroughly discussed in the literature. Concerning their visibility in X-ray plates there seems to be little accurate information. General opinion and clinical experience classify them as shadow-casting stones. Holmes and Ruggles, for example, have asserted that they are very dense. Thompson Walker in "Surgical Diseases of the Genito-urinary Organs," wrote that "oxalate of lime stones are the least permeable to the rays and throw the densest shadow; the rare cystin

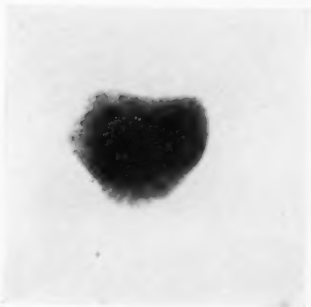

PLATE 1.—Above: A calculus of pure cystin. Drawing enlarged to twice actual size for the purpose of greater detail. Below: Röntgenogram of the stone after its removal. Its spongy texture is well shown.

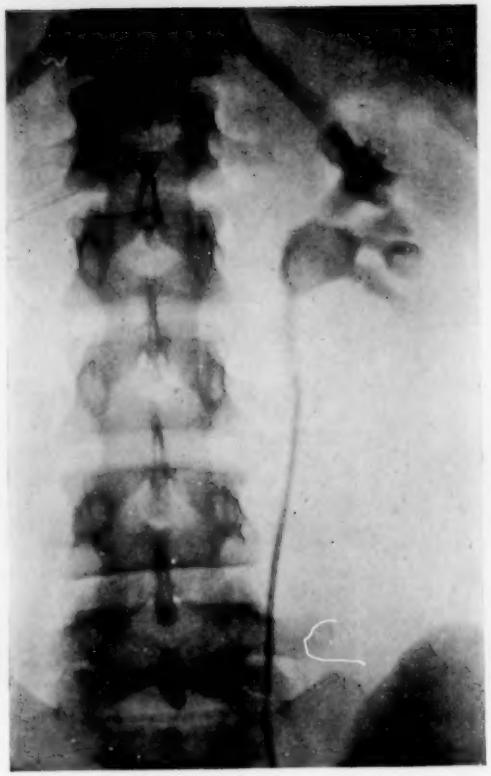

FIG. 1.—Case I. Pyelogram in which a shadowless stone appears as an area of lessened density, in the center of the renal pelvis.

FIG. 2.—Röntgenogram of the region of the stomach, made immediately after the taking of capsules containing bismuth and cystin. The former shows distinctly near the cardia. The encapsulated cystin could not be seen.

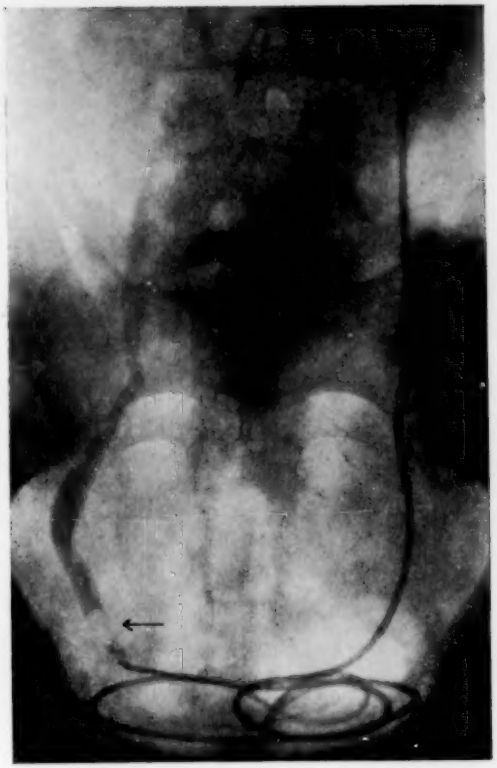

FIG. 3.—Case II. Ureterogram. The clear area in the line of injection beyond the catheter tip, marks the presence of the stone. Note the dilatation of the ureter above the level of obstruction.

and xanthin calculi throw a shadow slightly less dense; calcium phosphate is next....In an aseptic case the absence of a stone shadow after two or more examinations, when a plate of first quality has been obtained, excludes all but a pure uric acid calculus." In his "Practice of Urology," Chetwood stated that oxalate and phosphate stones "give a distinct image, and those of cystin, which are rare, also cast a faint shadow."

Clinical discussions concerning calculi of this type testify similarly that they are readily shown in X-ray plates. Morris, in 1906, wrote a paper devoted to the "X-ray Shadows of Cystic and Xanthic Oxide Calculi." The stones which he examined were from a museum collection. Of only one had there been a pre-operative radiogram. This stone had apparently cast a good shadow. It had been removed from the bladder of a boy of seven. It was of large size, but there is no complete description of its physical characteristics. In composition it was cystin with four-tenths per cent. inorganic residue. Müller, writing in 1911, recited at length the story of a young man of twenty-one who formed cystin stones with ungovernable persistence. Röntgenograms of both renal regions gave shadows of calculi on several occasions. Three nephrotomies and a litholapaxy were performed. The stones varied from millet-seed concretions to coral calculi of considerable size. The single chemical analysis given in this case reported, "cystin with a phosphate crust." From his observations Müller concluded that cystin stones are readily revealed by the X-ray. Wolf, Kienbock, and Neumann have also described their experiences with shadow-casting cystin calculi.

In spite of this unanimity of opinion, as expressed by radiologists, writers of text-books and clinicians, a pure cystin calculus in Case I, presented above, failed to reveal itself in two X-ray examinations. It should be borne in mind in this connection that cystin is an amino-acid, a normal constituent of protein. There would seem to be little reason to expect concretions composed solely of this substance to possess greater density than the body soft parts. For possible further evidence the following observations were made:

Two large gelatine capsules (Lilly's Size 000) were packed, one with bismuth subcarbonate (gm. 2.34), and the other with pure cystin (gm. 1.1). They were then coated with paraffin to keep them intact in the presence of the gastric secretion. A paraffin-covered gelatine capsule had been previously proved to be shadowless. The capsules were swallowed in quick succession, four and one-half hours after a light breakfast. Röntgenograms of the upper abdomen were made immediately, and again after about fifteen minutes. In both plates the control bismuth capsule was, of course, clearly visible and sharply defined. The cystin capsule could not be seen (Fig. 2).

The evidence at hand suggests that stones of pure cystin are shadowless. This statement should not be made, however, with too great finality. As noted by Arcelin in his excellent discussion, the opacity of a calculus is determined not alone by its composition, but by its thickness and structure as well. "The number of atoms making up a given thickness can modify considerably the

transparency of a stone, quite apart from the specific opacity of its elements." This is of fundamental importance. Because of differences in structure alone, therefore, it is possible for two stones of the same composition and thickness to present different degrees of permeability to the X-ray, one being of loose texture, and the other perhaps harder and more compact. Obviously it is not possible to judge definitely concerning the shadow-casting properties of any calculus without full knowledge of its size and structure, as well as of its chemistry. Even concretions of uric acid are not invariably shadowless. It is probably fair to say in general, however, that pure cystin calculi, with their characteristic loosely-knit structure, should be classified among the relatively invisible stones. Unusually compact structure, large size, or admixture with inorganic substances, may account for exceptions to this rule, and may explain some of the results of other observers.

Case II (Surgical No. 11159) is of less interest in this study. The röntgenogram, however, is strikingly similar to one presented by Stevens in 1917. A brief discussion of the case follows:

An American electrician, aged forty-five, entered the hospital complaining of pain in the left side. There was no family record of importance. The patient had had many of the childhood infections, among them scarlet fever in severe form at the age of six. At fifteen, he was acutely ill with what was thought to be "congestion of the kidneys." For several years before coming to the hospital he had complained of severe headaches.

Eight days before admission, he suffered a gradual onset of agonizing pain in the left lumbar region and flank. The attack was of brief duration and it subsided following the giving of an enema. There were repeated recurrences, however, and in the intervals there was persistent dull discomfort in the lower back upon the left side. The severe pains began in the lumbar region and extended forward into the flank and groin. On one occasion there was definite radiation to the genitalia. Nausea, vomiting, and headache accompanied the later attacks. There was no fever. The physician in attendance found no blood in the urine. Because of continued pain hospital entry was decided upon.

Examination found a well developed and fairly nourished man of middle years. He was nervous and apprehensive, but his appearance was not suggestive of acute illness. The temperature was very slightly elevated; pulse and respirations, normal. There was definite costo-vertebral tenderness on the left side. The routine physical examination was otherwise unimportant. The urine contained a slight trace of albumen and a small amount of pus and microscopic blood. The leucocyte count was 9000. X-ray studies of the urinary tract revealed no evidence of calculi. The left kidney was large and low.

Cystoscopy found a normal bladder. There was occasional slight bleeding, however, from the left ureteral orifice. The right ureter was readily catheterized and the right kidney was apparently normal. The left ureter presented definite obstruction at a point three cm. above the level of the bladder. X-ray plates were then made with the radiographic catheters *in situ*. There appeared at the tip of the left catheter a very small, faint area of opacity. It was circular in outline and, in its appearance, suggestive of the ordinary phlebolith. A more positive picture was furnished by the ureterogram in which again a stone was shown by negativity. Beyond the catheter tip, and interrupting the shadow of the filled ureter, was an oval vacuole of decreased density, about one cm. in length. Above this point there was well-marked ureteral dilatation (Fig. 3). The picture was

obviously produced by a relatively shadowless calculus displacing the opaque solution. The small shadow shown before injection, if a part of the stone, may have been its denser nucleus.

The passage of the calculus was not accomplished by intra-vesical measures and the patient refused operation. He was, therefore, discharged. In answer to a recent letter of inquiry he has informed us that he passed his stone several days after leaving the ward. Unfortunately we have no knowledge concerning its composition and physical characteristics. The calculus in the similar picture noted by Stevens was of the urate group.

CONCLUSIONS

It is apparent that, following the injection of an opaque solution into the ureter and renal pelvis, invisible stones may sometimes be shown by negativity.

Contrary to the general opinion, calculi of pure cystin should be classified in general with those concretions which possess no greater density than the body soft parts.

BIBLIOGRAPHY

[1] Arcelin: Encyclopédie Française d'Urologie. Paris, Octave Doin et Fils, 1914, vol. ii, pp. 156–177.

[2] Chetwood, C. H.: The Practice of Urology. New York, Wm. Wood and Co., 1916, p. 136.

[3] Cohn, J.: Verhandl. der Berl. med. Gesellsch., 1899, vol. xxx, pp. 188–189.

[4] Holmes and Ruggles: Röntgen Interpretation. Philadelphia, Lea and Febiger, 1919, pp. 192, 193.

[5] Kienbock: Wien. klin. Wchnschr., 1903, vol. xvi, p. 545.

[6] Lichtenstern: Wien. klin. Wchnschr., 1903, vol. xvi, pp. 544, 545.

[7] Morris, Henry: The Lancet, 1906, vol. ii, pp. 141, 142.

[8] Müller, Achilles: Wien. med. Wchnschr., 1911, vol. xxxvii, pp. 2364–2369. *Ibid.*, vol. xxxviii, pp. 2488–2492.

[9] Neumann, A.: Deutsch. med. Wchnschr., 1914, vol. xl, pp. 2065–2066.

[10] Stevens, A. R.: Jour. Am. Med. Assn., 1917, vol. lxviii, p. 1903.

[11] Thompson Walker, J. W.: Surgical Diseases and Injuries of the Genito-urinary Organs. New York, Funk and Wagnalls Co., 1914, p. 40.

[12] Wolf, J.: Verhandl. der Berl. med. Gesellsch., 1899, vol. xxx, p. 174.

INTRA-ABDOMINAL HEMORRHAGE FROM RUPTURED CORPUS LUTEUM*

By Edward C. Moore, M.D.
of Los Angeles, California

Intraperitoneal hemorrhage may arise from various sources, most often from a ruptured ectopic pregnancy. Aside from hemorrhages of a purely traumatic origin, such as ruptured liver, kidney, spleen or pancreas, spontaneous rupture of the uterus may be mentioned. Very rarely, exceedingly serious intra-abdominal hemorrhage occurs, traceable to none of these sources, but either to a ruptured graafian or atretic follicle or to a ruptured corpus luteum.

A survey of all the available literature on intra-abdominal hemorrhage from ruptured corpus luteum impressed me with the fact that in none of the cases reported, so far as we have been able to ascertain, was the correct diagnosis made before operation. The reason for this is quite apparent; intra-abdominal hemorrhage following rupture of the corpus luteum is a rare occurrence compared with the other pathologic conditions which present an almost identical picture, and it is only natural that the diagnosis which most often fits a condition should be the one made.

It would seem that hyperæmia with engorgement of the blood-vessels could occur more readily in the ovary than in other less vascular structures. Hence it is not surprising that hæmatomas of the ovary are often found. It is reasonable to assume that when the graafian follicle is ripe and its ovum is extruded, the particular spot where the break occurred is weaker for a time, at least, than other portions of the ovary which had not been ruptured recently. Benthin [2] claims that even after the formation of the corpus luteum these sites remain very thin and are separated from the peritoneal space by an easily breakable layer of connective tissue.

It has been quite definitely proven that trauma plays a considerable part in the production of ovarian hemorrhage. Novak [8] quotes von Beust as having found nine cases out of thirty-six where trauma was considered the etiologic factor in producing the hemorrhage. He mentions one case occurring during a dance, one the result of a misstep and one due to intrapartum compression of an ovary. Several authors, Rubin,[12] Novak,[8] mention having cysts rupture during bimanual examinations, especially under ether, and at the laparotomy which followed having found the collapsed cyst with bloody fluid in the pelvis. Primrose [11] reports two cases; one patient was apparently perfectly well until she lifted a heavy box, whereupon she felt a sudden, sharp pain in her abdomen; the other patient's condition was complicated by acute appendicitis. Both women were taken violently ill two days before men-

* Read by title May 19, 1921, at the Meeting of Ex-resident Physicians of the Mayo Clinic.

struation was due. In the first case, Primrose found active hemorrhage coming from a ruptured corpus luteum, and in the second from a ruptured graafian follicle. Primrose argues that the strain from lifting the heavy chest caused the rupture in the first case, and that in the second case strain from vomiting during an attack of acute appendicitis ruptured the graafian follicle. Dansey's patient was seized with acute abdominal pain following straining at stool. The peritoneal cavity was found to be full of bright colored blood and both ovaries showed recent corpora lutea with hemorrhages.

It seems reasonable to conclude that any factor which excessively increases the hyperæmia of an ovary can produce ovarian hemorrhage. Whether this hemorrhage becomes intra-abdominal or intra-ovarian depends largely upon the point of least resistance, the weakest spot being the one to give way.

REPORTS OF CASES

CASE I.—Mrs. A. L., aged twenty-nine years, came for consultation December 27, 1915. She complained of dull, aching pains low in the abdomen, principally on the left side, which had begun about two weeks before. The pain continued intermittently until three days before examination, when she was suddenly seized with knife-like pains, without radiation, in the same location, so severe that they doubled her up. The pain lasted about fifteen minutes, and after it subsided she was weak and her side was sore. The following night the attack was repeated, but it was not so severe. There was no nausea, vomiting, fever or vesical disturbance. Menstruation had always been regular, a moderate flow for four or five days, until the two last periods, which were five days late, with slight flow of only two days' duration. There was no pain. She developed morning sickness, her breasts became large and tender, and she thought she was pregnant. She had had one child.

General physical examination revealed soreness and tenderness in the left lower quadrant of the abdomen. Pelvic examination made under ether disclosed the presence of an indefinite mass in the left side. A diagnosis of extra-uterine pregnancy was made.

At operation the following day the pelvis contained free and clotted blood. The uterus was slightly enlarged. The left tube and ovary and the right tube were normal. The right ovary, however, was somewhat enlarged and had a small clot protruding from a ruptured corpus luteum. This was resected and the ovary sutured. The patient recovered uneventfully.

Microscopic examination of sections of the ovary showed a typical corpus luteum which had ruptured.

CASE II.—Mrs. B. C. W., aged thirty-seven years, had been married twice. The first marriage was at the age of twenty-seven, and the second three months before examination. She had matured at the age of thirteen with regular menstruations of the twenty-eight-day type. The last period was on June 6, 1920. She had never been pregnant. She appeared to be perfectly healthy, except that she was troubled with constipation. She ate and slept well. June 27, 1920, she was feeling absolutely well at ten in the morning and was preparing to go

on an outing, when she was suddenly seized with severe, excruciating pain around the navel which gradually increased, and radiated to the lower costal margins. After taking some magnesia she felt nauseated and vomited.

The patient was seen by Dr. Leo A. Schroeder at 4.30 P.M. Her temperature was 98.7°, her pulse was 84. The systolic blood-pressure was 120, and the diastolic was 90. There was no abdominal distention and no rigidity of recti. At 7.30 P.M. I was called in consultation. At this time noticeable rigidity of both recti was present with marked tenderness over McBurney's point. Palpation caused increased pain in the epigastrium. The abdomen was otherwise negative. We were

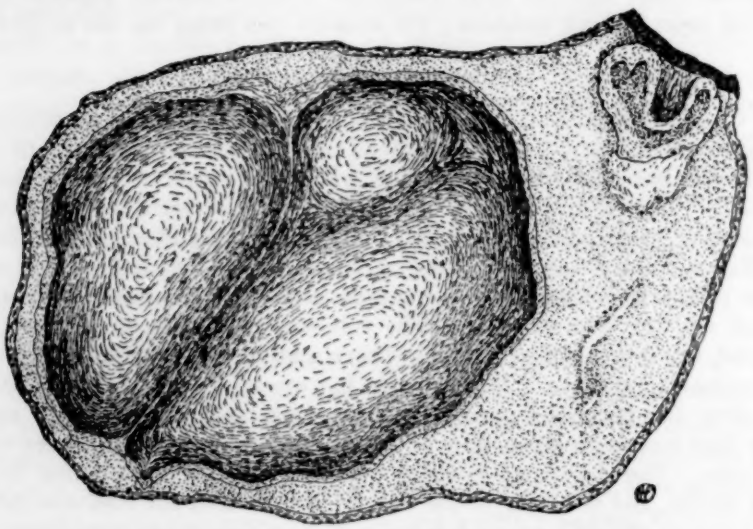

FIG. 1.—Sectioned ovary with ruptured corpus luteum.

so confident that this was a case of appendicitis that, like nearly all the surgeons whose reports have been tabulated, we did not make a blood count.

At 9 P.M. we operated through a McBurney incision. On opening the peritoneum, the abdominal cavity was found filled with blood. The incision was immediately extended downward so as to expose the uterus and adnexa. The hemorrhage was discovered on the upper posterior surface of the right ovary and looked as though it were coming from a normal corpus luteum. The ovary was clamped, and the remaining abdominal organs were examined, all of which, including the appendix, appeared to be normal. The right ovary was again examined and the clamp removed in order to verify our former observations and to prove to our satisfaction that this was the source of bleeding. Bleeding began immediately, not an ooze, but rather a brisk hemorrhage. The greater part of the ovary was removed; the stump left was 0.6 cm. wide. After the abdominal cavity had been filled with normal salt solution the incision was closed in the usual way. The patient made a normal recovery and left the hospital on the tenth day.

Pathologist's report: The specimen is a cystic ovary hardened in formalin. It measures 3.0 by 3.5 by 2 cm. Attached to the upper pole is a firm clot 1 cm. in diameter. On section, immediately beneath the blood clot, there is a corpus luteum about 1 cm. in diameter. (See Fig. 1.) A cyst filled with a gelatinous substance occupies about three-fourths of the ovary. Microscopic sections show a typical corpus luteum which has ruptured.

COMMENT.—In order better to study these cases a tabulation was made of those it was possible to obtain from the literature. The author's own words have been used whenever space permits. The list is smaller than it would be if I had had access to more of the literature on this subject since I included data of only the original articles which I had read. (See Table.)

In general the reports show that the symptomatology of this condition is characterized by sudden abdominal pain, cramplike or colicky, followed almost always by nausea and vomiting. There is nothing characteristic about the temperature and pulse. Localized abdominal pain and tenderness, generally on the right side, are noted almost immediately after the onset of the sudden pain. Frequently abdominal rigidity and distention are present. The patient is pale, but seldom seems to be in shock. While these symptoms in a woman are characteristic of trouble in either the appendix or fallopian tube, the possibility of a hemorrhage from the ovary must be considered, especially if the patient shows a marked degree of pallor or anæmia.

There was practically no blood work done, or if done, it was not reported. I believe that a knowledge of the percentage of hæmoglobin, or a complete blood count, in the two cases reported herewith, might have helped in making a better diagnosis, particularly in the second case. Marital relations do not seem to play a part, since quite a number of the hemorrhages occurred in young unmarried women.

Speese [13] believes most such hemorrhages occur during or within a few days of the menstrual period. In Bookman's [3] case hemorrhage occurred seven days after normal menstruation and in Warnshuis' [14] case fourteen days after. In Adams' [1] case the patient expected to menstruate at any time, and after suffering for three days from acute abdominal pain, vomiting and diarrhœa, normal menstruation began and lasted five days. On the following day, which was the ninth of her illness, she was operated on and the abdomen was found to contain three pints of blood-stained fluid and clots. In my second case the patient was taken ill seven days before her period was due. Conclusions cannot be drawn from this small number of cases as to the rôle which the menstrual function plays in these hemorrhages. Neither does it seem to determine the kind of follicle to be ruptured in case hemorrhage occurs, this being demonstrated by the two cases reported by Primrose.[11] In his first case the hemorrhage was due to a ruptured corpus luteum and in the second case to a ruptured graafian follicle, yet in both cases the

CASES REVIEWED OF INTRA-ABDOMINAL HEMORRHAGE FROM RUPTURED CORPUS LUTEUM

Author	Age	Symptoms	Abdominal pain and tenderness	Nausea and vomiting	Temperature and pulse	Abdominal distention and rigidity	Sudden increase in abdominal pressure	Pallor	Blood findings	Normal menstrual history	Married	Pregnancy	Diagnosis before operation	Free blood in the abdomen	Ruptured corpus luteum	Complicated with appendicitis
Hind (1905)			Sudden pain		Patient pulseless, unconscious			*		*	*	0		Several pints of clotted blood	Left ovary	
Ladinski (1910)	22	Not mentioned	Sharpattacks of pain; tenderness most marked in right inguinal region			Moderate distention							Tubal pregnancy	Large amount of fluid and large clots	Left ovary	
Primrose (1912)	35	Two days before menstruation	Sudden severe pain tenderness general; most marked in left iliac and hypogastric regions	Nausea; frequent vomiting	97.6 130	Marked distention; rigidity	Accidental strain while lifting heavy chest	*		*	*	3½ years before		Large amount; clots	Left ovary	0
Primrose	40	Two days before menstruation	Acute pain; tenderness in right iliac region	Violent attack of vomiting	88.6 80	Rigidity	Accidental strain while vomiting			*	*	0	Appendicitis	Large amount; dark blood clots	Ruptured graafian follicle R + Ov.	Acute septic

Warnshuis (1912)	17	Fourteen days after menstruation	Severe pain; tenderness severe over appendix	Nausea	99.4 84	Distention; rigidity		*		Regular with pain and clots	o		Appendicitis	Bright red with clots	Left ovary	Chronic
Adams (1913)	18	Three days before menstruation	Acute pain; tenderness in iliac fossas, particularly marked on right side	Vomiting		Rigidity					*	o	Appendicitis	About 3 pints free blood; fluid and clots	Left ovary	*
Bookman (1914)	19	Seven days after menstruation	Sudden, severe, cramp-like pain	Vomiting	101.8 100	Muscular resistance		*			o		Appendicitis	Bright red blood and blood clots	Right ovary	*
Lipscomb (1916)	25	Not mentioned	Sudden, colicky pain; tenderness particularly in right iliac fossa	Vomiting	99.4 100			*			*	o	Appendicitis	One quart	Left ovary	o
Dansey (1916)	21	Not mentioned	Sudden, acute, colicky pain; tenderness acute in right iliac region		100.8 120		From straining at stool	*			o		Appendicitis	Peritoneal cavity full of bright blood	Right and left ovaries	o
Browne (1916)	22	Not mentioned	Tenderness in right iliac fossa	Profuse vomitus	High fever						o		Appendicitis	Filled with thin, dark blood	Left ovary	*

CASES REVIEWED OF INTRA-ABDOMINAL HEMORRHAGE FROM RUPTURED CORPUS LUTEUM—(*Continued*)

Author	Age	Symptoms	Abdominal pain and tenderness	Nausea and vomiting	Temperature and pulse	Abdominal distention and rigidity	Sudden increase in abdominal pressure	Pallor	Blood findings	Normal menstrual history	Married	Pregnancy	Diagnosis before operation	Free blood in the abdomen	Ruptured corpus luteum	Complicated with appendicitis
Novak (1917)	15	Ten days after menstruation	Sudden pain, violent in right iliac fossa	Nausea; vomiting	100.2 138	Rigidity of right rectus muscle		*	Hemoglobin 45 per cent.; leucocytes 20,000; polymorphonuclears, 90 per cent.	*	0	Left tubal pregnancy, nonruptured	Appendicitis	Large quantity	Ruptured follicular cyst, right ovary	0
Speese (1920)	20	Six days before menstruation	Sudden, severe pain	Vomiting			No trauma or strain	*	Leucocytes 18,850	*	*	0	Appendicitis	Much fresh blood and large clots	Right ovary	Chronic
Moore	29	During menstruation	Suddenknife-like pain in left lower abdomen; tenderness			No distention or rigidity				Regular, normal until last two months	*	1	Tubal pregnancy	Large amount	Right ovary	0
Moore	37	Seven days before menstruation	Sudden pain, excruciating about navel; 7.30 p. m. marked tenderness over McBurney's point	Nausea and vomiting after taking magnesia	98.7 84	4.30 p. m. no distention or rigidity; 7.30 p. m. rigidity				*	*	0	Appendicitis	Entire abdominal cavity filled with bright red blood	Right ovary	0

hemorrhage occurred two days before menstruation was due. His second case really should not be included in the tabulation, but it was used in order to draw especial attention to this interesting point.

If the menstrual history has been normal and if the patient has had previous intestinal disturbances simulating appendicitis, it is quite natural that a preoperative diagnosis of acute appendicitis should be made. This was the diagnosis made in my second case, while in the first, in which menstruation had been irregular, a diagnosis of ruptured ectopic pregnancy was made. As soon as the abdomen is opened and it is found to be filled with blood, the surgeon decides that the case is a ruptured tubal pregnancy. A rapid search, usually through a right rectus incision, fails to locate bleeding from either tube. Further examination shows the bleeding to be coming from the ovary. Sometimes the condition is complicated by the presence of acute or chronic appendicitis, but often the appendix is found to be normal.

Six of the fourteen tabulated cases were complicated by appendicitis. There may be some association between the two conditions, but what that association is has not been determined. It may be, as suggested by Primrose, that the follicle is ruptured by strain produced by a violent attack of vomiting.

The ovary should be considered an aggregation of follicles, some maturing and some retrogressing, constantly changing their appearance and function from day to day. The structures surrounding the different follicles also change, depending on the development and special function of the follicles. The ovarian stroma with its nerves and blood-vessels forms the framework for housing these follicles. Each follicle seems to have a life cycle of its own. All appear alike during their early development, but each month one gradually outstrips the others in growth and develops into a graafian follicle. Whether this follicle was always different from the others, or whether because of its richer blood supply it grows faster, is not known. At any rate, the ovum of this largest follicle, called the graafian follicle, is the only one which matures and when ripe is extruded. As soon as this takes place, there is no further growth of the other follicles which started to mature; their ova die and gradually disappear, leaving the so-called atretic follicles. Only one ovum goes to maturity each month. Following the giving off of the ovum great changes occur in the graafian follicle, producing what is known as the corpus luteum.

The blood supply of the graafian follicle and of the corpus luteum has been worked out so thoroughly by Novak,[9] from a study of a large number of ovarian sections, that my statements have been obtained from his writings. The maturing follicle, it appears, receives its blood supply from vessels in the theca interna, from which tiny offshoots penetrate into the granulosa. The earliest stages of the corpus luteum are marked by a great increase in the number and size of these vessels in the theca and also at the base of the granulosa. This fact is of particular interest, since it seems to indicate that, due to the stage of hyperæmia which follows the extrusion of the ovum,

hemorrhage can occur more readily into a corpus luteum than into a graafian follicle.

To the physician who has not actually seen an abdomen filled with blood exactly as it is found during a ruptured ectopic pregnancy, it does not seem possible that such extensive hemorrhage could come from the tiny vessels which encircle the follicles. That this is actually what does occur occasionally has been observed sufficiently often by a number of surgeons, who have not only seen the hemorrhage coming from a ruptured ovary, but also have removed either the entire ovary or a portion of it, to prove microscopically that the bleeding originated either from a ruptured graafian or atretic follicle or from a ruptured corpus luteum.

Penny's [10] case proves that hemorrhage of this kind can terminate fatally. His patient, a healthy woman, mother of two children, was taken ill at 7 P.M. She was menstruating at the time and attributed her symptoms to this fact. She gradually grew worse and died shortly before 4 A.M., having been ill less than nine hours. Necropsy showed a large amount of dark fluid blood in the abdominal cavity, the right side especially being full of fluid and semicoagulated blood. In the right iliac fossa was a rather firm blood clot which led to the right ovary to which a firm clot 2.5 cm. long was attached. There were two ruptured graafian follicles, to one of which an ovum was adherent.

BIBLIOGRAPHY

[1] Adams, F.: Hemorrhage from a Corpus Luteum Cyst. Brit. M. J., Lond., 1913, No. 2727, 712.

[2] Benthin, W.: Hämatocele als Folge einer geplatzten Corpusluteum Cyste. Monatschr. f. Geburtsh. u. Gynaek., Berl., 1913, xxxvii, 532-534.

[3] Bookman, M. R.: Report on a Case of Rupture of Corpus Luteum with Intraperitoneal Hemorrhage. Am. J. Obst., N. Y., 1914, lxx, 416-420.

[4] Hind, W.: Sudden Hemorrhage from the Ovary. Brit. Med. J., Lond., 1905, ii, 189.

[5] Ladinski, L. J.: Bleeding from Corpus Luteum. Am. J. Obst., N. Y., 1910, lxii, 487.

[6] Lipscomb, T. W., Dansey, St. J. W. and Browne, H.: Three Cases of an Acute Abdominal Affection Due to Hemorrhage from a Ruptured Corpus Luteum. Med. J. Australia, Sydney, 1916, ii, 57-58.

[7] Novak, E.: The Corpus Luteum, Its Life Cycle and Its Rôle in Menstrual Disorders. J. A. M. A., Chicago, 1916, lxvii, 1285-1291.

[8] Novak, E.: Abdominal Hemorrhage of Ovarian Origin. J. A. M. A., Chicago, 1917, lxviii, 1160-1162.

[9] Novak, E.: Hæmatomata of the Ovary, Including Corpus Luteum Cysts. Johns Hopkins Hosp. Bull., Balt., 1917, xxviii, 349-354.

[10] Penny, E. J.: Case of Fatal Hemorrhage into the Abdominal Cavity during Menstruation. Ann. Surg., 1886, Phila., iv, 456-457.

[11] Primrose, A.: Hemorrhage into the Peritoneal Cavity Caused by Accidental Rupture of the Ovary. Ann. Surg., Phila., 1912, lvi, 125-133.

[12] Rubin, I. C.: The Simulation of Corpus Luteum and Retention Cysts of the Ovary with Ectopic Pregnancy and Early Uterine Abortion. Surg., Gynec. and Obst., Chicago, 1917, xxiv, 443-454.

[13] Speese, J.: Intra-abdominal Hemorrhage from Ruptured Corpus Luteum. Ann. S., Phila., 1920, lxxi, 777-778.

[14] Warnshuis, F. C.: A Report of a Case of Ruptured Ovary, with Large Intra-abdominal Hemorrhage and Symptoms of Acute Recurrent Appendicitis. Phys. and Surg., Ann Arbor and Detroit, 1912, xxxiv, 123-127.

PREMATURE OSSIFICATION AFTER SEPARATION OF THE LOWER RADIAL EPIPHYSIS

By Morris K. Smith, M.D.
of New York, N. Y.

T. B., a boy of seventeen, applied at the Out-patient Department of St. Luke's Hospital in May, 1919, with a traumatic separation of the left lower radial epiphysis, at first thought to be a Colles's fracture. There was backward displacement of the epiphysis carrying with it a thin shell of bone from the posterior surface of the diaphysis, as so often happens in this type of injury (Fig. 1). The ulnar styloid was also broken. The displacement was treated by reduction under gas anæsthesia and the patient ultimately discharged, the result being considered fair. About eight months later he returned complaining of prominence of the head of the ulna on the injured side and weakness and pain in the wrist. An X-ray made at this time showed some obliteration of the radial epiphysial line (Fig. 2). At the end of a year from the date of injury further X-rays were made. At this time there was complete ossification of the radial epiphysis while that of the ulna had largely disappeared (Fig. 3).

At the most recent examination, over two years from the time of injury, the patient stated that he had good use of his wrist. The head of the ulna was prominent. The radius measured three-quarters inch less than that on the uninjured side and the total length of the forearm from olecranon to tip of middle finger was three-quarters inch less than its fellow. There was no difference in the length of the two ulnæ.

The interest in this case lies in the clear radiographic demonstration of premature ossification of the previously separated epiphysis, resulting in arrested growth. The process in the radius was observable in eight months and complete in one year. Another feature of interest is the accompanying ossification in the ulna epiphysis, although the original injury here was a fractured styloid.

Traumatic separation of the epiphysis is a fairly frequent type of injury and may be mistaken for ordinary fracture. The prognosis should be guarded in all such cases on account of the possibility of arrested growth. It is impossible to tell from the clinical and radiographic evidence of the injury when it is to be expected. It has occurred when there has been no displacement.

I have not been able to find any data as to the exact frequency of arrested growth after epiphysial separation beyond the fact that it is rare. Poland [1] in his exhaustive study collected fifty-six cases of arrest of development, of which seventeen were at the lower end of the radius. Stimson [2] states that a few such cases have been reported and mentions two personally observed

in which injury at the lower end of the radius at the age of fourteen years produced a late deformity exactly resembling that of a bad Colles's fracture. Pilcher [3] reported a case of arrested growth in a man whose wrist he had originally reduced twenty years before. Tanton [4] described a case of arrested growth at the lower end of the radius and quoted Walter, Chaput and Lorenz as having reported similar cases. Others who have mentioned such cases include Andrews [5] and Murphy.[6]

SUMMARY

Traumatic separations of the epiphyses are not uncommon, among the most frequent being that of the lower end of the radius. Arrest of development following this injury sometimes occurs, although reported cases are not numerous. In the case described the premature ossification was evident radiographically in eight months and complete in one year.

REFERENCES

[1] Poland, J.: Traumatic Separation of the Epiphyses. London, Smith, Elder & Co., 1898, 107.

[2] Stimson, L. A.: Fractures and Dislocations, 8th ed. New York and Philadelphia, Lea & Febiger, 1917, 30.

[3] Pilcher, L. S.: Fractures of the Lower Extremity or Base of the Radius. Philadelphia and London, J. B. Lippincott Co., 1917, 45.

[4] Tanton, J.: Bulletins et Mémoirs de la Société de Chirurgie de Paris, 1915, 412, 2267.

[5] Andrews, E. W.: ANNALS OF SURGERY, 1902, 35, 663.

[6] Murphy, J. K.: Translation of a Handbook of the Surgery of Children, by E. Kirmisson. Oxford University Press, Henry Frowde: London, Hodder & Stoughton, 1910, 301.

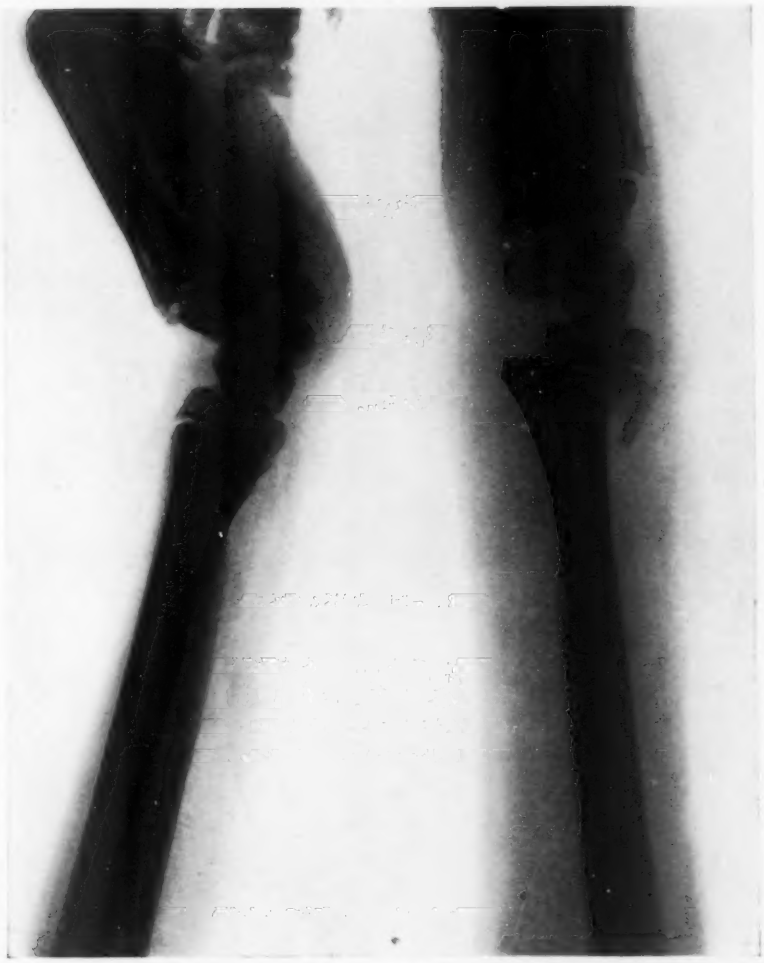

FIG. 1.—Traumatic separation of lower radial epiphysis carrying with it a thin shell of bone from the posterior surface of the diaphysis.

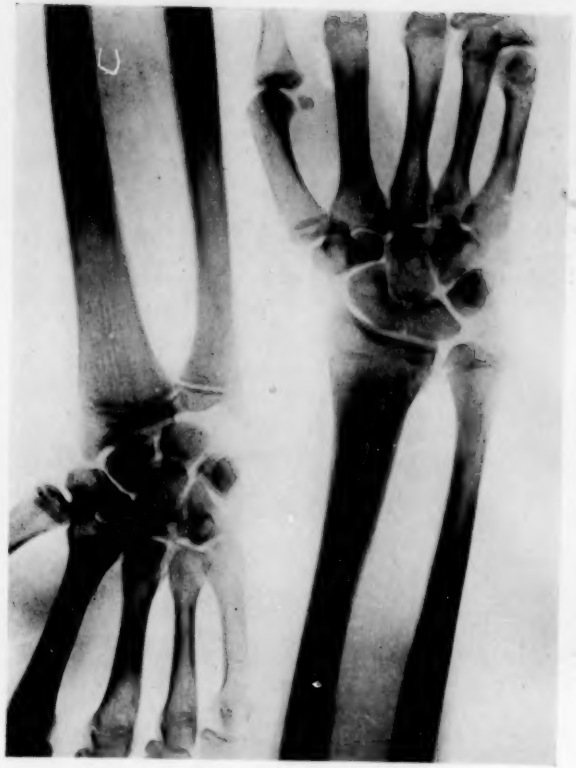

FIG. 2.—Eight months after injury, there is partial obliteration of radial epiphyseal line.

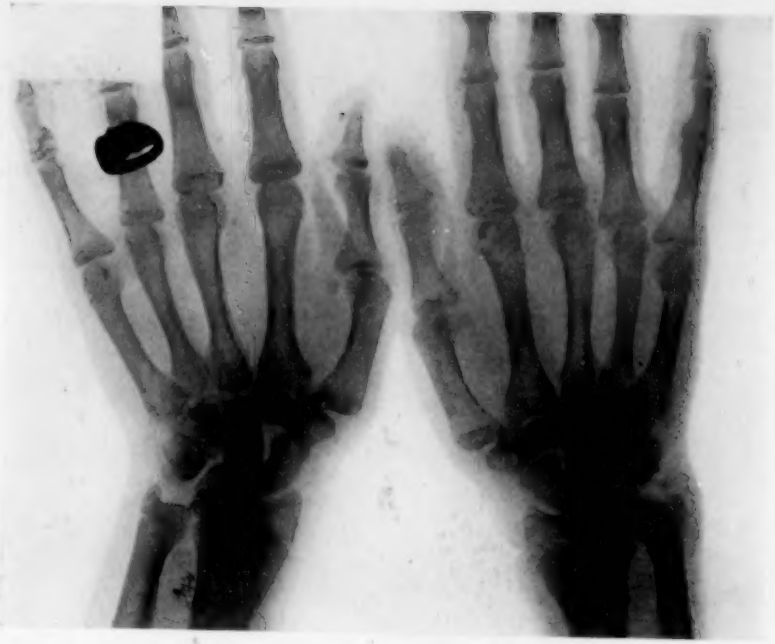

FIG. 3.—One year after injury. Lower radial epiphysis is completely ossified while that of the ulna is largely so.

TRANSACTIONS

OF THE

PHILADELPHIA ACADEMY OF SURGERY

Joint Meeting with the New York Surgical Society held November 7, 1921

The President, Dr. George G. Ross, in the Chair

DRAINAGE

Dr. Joseph A. Blake, of New York, read a paper with the above title, for which see page 385.

THE PRESENT STATUS OF EPIPLOPEXY

Drs. John H. Gibbon and John B. Flick, of Philadelphia, read a paper with the above title, for which see page 449.

REGENERATION OF LOWER JAW

Dr. Robert H. Ivy presented a little girl, aged seven, who was first seen in January, 1921, suffering from osteomyelitis of the lower jaw originating in dental infection, with complete sequestration extending from the first permanent molar on the right side to the canine region of the left side. Removal of the sequestrum was required, leaving a separation of about an inch and a half between the remaining portions of the mandible. Doctor Ivy showed her especially to demonstrate the complete regeneration of bone which had occurred since that time. The X-ray in October showed complete regeneration of the bone and restoration of continuity. There was also exhibited a wire splint made by Dr. John Ross, cemented to the molar teeth on each side, by which the full width of the lateral halves of the jaw was preserved during the process of regeneration, with resulting excellent occlusal relationship between the remaining lower and upper teeth.

CICATRICIAL ANKYLOSIS OF THE JAW

Dr. John H. Jopson and Dr. Robert H. Ivy presented a colored girl, aged twenty-three, who at the age of eight, she states, had typhoid fever which caused ulceration of the cheek and resulted in adhesion of the inside of the cheek to the lower jaw, producing inability to separate the upper and lower teeth. She came to the Polyclinic Hospital the beginning of September with inability to separate the incisor teeth more than one mm. A knife blade could not be passed through. Wassermann reaction was negative although she said she had received injections for "bad blood" two or three years previously. At operation it was found that adhesions held the mucous membrane of the cheek to the lower jaw. By cutting the adhesions back as far as the ascending ramus it became possible to get the jaw open about three cm.

The jaw was kept blocked open with a wooden wedge attached with wire between the teeth on the left side. To cover the raw surface of the inside of the cheek, a flap of skin was raised from the right side of the neck with its pedicle about the region of the mental foramen and extended back toward the mastoid process. This flap was inserted into the mouth through a buttonhole incision in the lower vestibule and sutured to cover as much of the raw surface as possible. A few weeks later under local anæsthesia the connection with the outside skin was severed. While the result is not perfect and there is some recurrence of the contraction, the patient is now able to masticate solid food. The operation is being supplemented by the use of a home-made wooden wedge which the patient uses herself.

END RESULTS OF OPERATION FOR DUPUYTREN'S CONTRACTURE

Dr. Arthur Bruce Gill said that in the Annals of Surgery of August, 1919, he published a description of a method of operation for the cure of Dupuytren's contracture and reported one case operated upon by the method described.

Briefly, this operation consists of: first, an incision in the palm along the line of the distal palmar crease; second, a complete excision of the palmar fascia from the webs of the fingers to the base of the palm through this incision; third, the insertion beneath the skin of a free fat transplant from the thigh. If the fascia on the palmar aspect of the proximal phalanges is contractured it should be excised through transverse incisions along the creases at the bases of the fingers. If the proximal interphalangeal joint of a finger cannot be extended after such excision of fascia the head of the proximal phalanx should be excised through a transverse posterior incision over the joint.

The case which he reported at that time he now presented again. The operation on his hand was performed three years ago. He has worked steadily ever since. The scar of the incision is almost invisible, the palm of the hand is soft and normal to appearance and touch, and there has been absolutely no return of the former contracture. But it is interesting to observe that there is some contracture of the fascia of the thenar eminence. This portion of the palmar fascia was not excised at the operation because it was not then contractured. It should now be removed through an incision following the crease around the base of the thumb.

He then presented two other cases:

Case II.—W. J. He was operated on for Dupuytren's contracture of the left hand in October, 1919. Contracture had been present for five years. The little finger was flexed tightly into the palm. The ring and middle fingers were moderately flexed. The operation was performed as described. It included excision of the fascia on the palmar aspect of the fingers and the excision of the head of the proximal phalanx of the little finger. The skin of the palm of the hand along the line of the distal palmar crease was considerably macerated

owing to the long-continued extreme contracture. Because of this condition the wound opened up slightly after the operation. There was serous discharge for a period of three or four weeks, but no sloughing of the fat transplant. As in case number one the result is entirely satisfactory; the scar is scarcely visible, the hand is absolutely free of contracture, the palm is soft and pliable. The little finger is straight but there is a fibrous ankylosis at the proximal interphalangeal joint. This, however, does not interfere in the least with his usual work as a laboring man, whereas the contracture of the finger formerly prevented his holding tools and annoyed him greatly.

CASE III.—J. B. This patient is presented through the courtesy of Dr. A. P. C. Ashhurst, under whose care he has been. In October, 1916, both hands were operated upon by the Adams method of multiple subcutaneous tenotomies of all the contractured bands of fascia. At that time he was thirty-nine years of age. For eight months he had had Dupuytren's contracture of both hands, the third, fourth and fifth fingers being involved, and the fifth finger being flexed completely into the palm. He was employed in a lead factory and his fingers were constantly flexed when at work.

Following the operation he wore gypsum splints continuously for six weeks and then only at night for six weeks more. Following this he had massage three times a week for several weeks. The left hand was improved by the operation while the right hand was not so useful as it had been before. In the latter the only use was of the thumb and index finger. The other fingers remained still in extension. In the left hand the third, fourth and fifth fingers had slight active flexion. In November of 1920 his left hand had relapsed and become very much worse even than the right. November 23, 1920, operation was done by Doctor Ashhurst after the method which the speaker had advocated. At the end of a year both his hands are free of contractures, palmar tissues are soft and pliable, and the scars are scarcely apparent. The interphalangeal joints, however, are more or less rigid. This latter condition illustrates one of the disadvantages of the Adams procedure. The splinting which is necessary after the subcutaneous tenotomies not infrequently leads to fibrous ankylosis of the fingers. After the operation now described a palmar splint is applied, which extends only to the base of the fingers, for a week or ten days. This allows free use of the fingers during this period. As soon as the hand is healed the patient is allowed to return to his usual work.

It is evident that the operation in these three cases has been eminently satisfactory. It seems reasonable to believe that in this method of operation we have a complete solution of this troublesome condition. The fibrous ankylosis of the finger in case two he had seen occur in other instances following the excision of a portion of the phalanx, to relieve a long-continued contracture of the interphalangeal joint. In one case of ankylosis of the finger he had inserted a free fat transplant following the excision of a portion of the phalanx. This case when last seen had motion present in the joint.

Dr. John H. Gibbon stated that since hearing Doctor Gill's first report on this condition some time ago, he had operated upon a case by this method with most satisfactory results. He believed that the old method with the longitudinal incisions is not comparable to this.

Dr. James T. Rugh reported that he had had two more cases of the same type, operated on in the same manner with excellent results, far superior to the old time operation.

Stated Meeting Held December 5, 1921

The President, Dr. George G. Ross, in the Chair

LANTERN DEMONSTRATION OF CYSTIC DISEASES OF BONE AND BONE TUMORS

Dr. Ralph S. Bromer (by invitation) presented a series of lantern slides illustrating the röntgenologic diagnosis of cystic diseases of bone and bone tumors.

Dr. Astley P. C. Ashhurst presented a series of lantern slides illustrating the clinical diagnosis, prognosis and treatment of cystic diseases of bone and bone tumors.

Dr. C. Y. White (by invitation) presented a series of lantern slides showing microphotographs of cystic diseases of bone sarcoma and discussed the pathological diagnosis.

THE CAUSE OF DEATH IN HIGH INTESTINAL OBSTRUCTION

Dr. J. W. Ellis, of the Medical Corps of the U. S. Navy, read a paper with the above title, for which see page 429.

EXTERNAL BILIARY FISTULA

Dr. John H. Jopson and Dr. John Speese reported a case of obstruction of the common bile duct with complete external biliary fistula, relieved by choledochogastrostomy.

The patient, a male about forty-five years old, was admitted to the Presbyterian Hospital in October, 1920, suffering from an acute upper abdominal inflammation of one week's duration, accompanied by chills, fever and deep jaundice. Two days later he was operated upon by Doctor Speese, and a perforated gall-bladder containing a number of small stones was drained. Bile and calculi were found outside the gall-bladder. A biliary fistula persisted, and six weeks later a second operation was performed to effect its closure. No stones had been found in the common duct at the first operation, but the stools remained clay colored and an obstruction was evidently present. At the second operation, which was very difficult by reason of the extensive adhesions present, the gall-bladder was found to be almost entirely sloughed away. With difficulty the common duct was located, and it was thought that a probe could be passed into the duodenum. A tube was inserted into the duct, and the wound was partially closed. No improvement resulted, and four weeks later another operation for the relief of the obstruction and cure of the fistula was attempted. The same difficulties were

encountered in the recognition of structures, due to the dense and massive adhesions. When the duct was uncovered it was found to be obliterated in its lower portion. It seemed impossible to expose the duodenum sufficiently to establish a communication with the duct, and as the pylorus was accessible and in close proximity, a fistula between it and the upper portion of the common duct was constructed. A piece of Dakin tubing was passed into a lateral opening in the duct, sutured to its margin, and the other end was introduced into the stomach, the walls of which were tacked to the duct by sutures in front of and behind the opening. The technic was similar to that sometimes employed in choledochoduodenostomy. Leakage of bile in diminishing amount persisted from the wound for a time, soon became scanty, and after several weeks the wound closed, and has remained healed. Bile reappeared in the stools after twelve days. For a time the patient had recurrent attacks of pain and jaundice, evidently associated with cholangitis, but these have ceased, and he is now in good condition.

The operation of anastomosing the common duct to the duodenum has been performed many times, where obstruction was present which could not be removed; also where reconstruction by plastic operation was impossible in cases where the duct had been accidentally wounded or multilated, or where it had been deliberately resected. In the latter cases an external biliary fistula, with its attendant disabilities and dangers, threatens or is already established. While the operation may be difficult, involving as it does the exposure of the upper portion of the common duct, or of the hepatic duct, in a mass of adhesions in the old cases, and its approximation and suture to the near-lying duodenum, such terminal or lateral approximation can usually be accomplished. In this case operated by us, it was impracticable by reason of the adhesions burying the duodenum, and the anastomosis to the pyloric end of the stomach proved eminently satisfactory.*

LYMPHOSARCOMA

Dr. J. Ralston Wells, from the service of W. Estell Lee, M.D., at the Children's Hospital, Philadelphia, Pa., reported the history of a boy, nine and one-half years of age, who was admitted to the hospital August 8, 1921, on account of a lump in his left axilla. Approximately one year ago he began to have boils over the body and arms. No history of any specially chronic ones at seat of present lump (mass). About seven months ago first noticed a swelling in the left axilla. At this time it was about the size of an ordinary marble. This lump, as time went on, gradually increased in size, although without treatment at times seemed to become smaller for a week or so and again resume its progressive enlargement. Three weeks ago two spots appeared near the apex of the rounded mass and showed a tendency to ulcerate. During the last

* Since reporting this case Dr. Ellsworth Eliot has called their attention to the statistics collected by him in his article "The Repair and Reconstruction of the Hepatic and Common Bile Ducts," read before The American Surgical Association in 1917, in which six cases are mentioned as treated by this method of choledochogastrostomy, at least five of which were successful.

week the lump has increased very rapidly in size, one of the above-mentioned spots has broken the skin, forming an ulcer which has a serous, slightly odorous discharge. A few darting pains have been present in the mass during the last few weeks. Patient unable to define the direction the pains radiated. Pains have never been severe or prolonged over several seconds. Usual diseases of early childhood, otherwise has been " very well." Family are well, no history of tuberculosis or neoplasm. The patient is a white boy. Well developed, well nourished, good color, and apparently in good health. Facial expression good; skin of good color and texture, warm and normally moist. No rash or other abnormal qualities noted.

The left anterior axillary, upper part, is the seat of a relatively large indurated swelling approximately six cm. in diameter at the base, and four cm. from base to apex (apices). This mass terminates in two tit-like formations, dark purple in color, one is ulcerated, the other apparently contains fluid, two to three c.c., and is apparently covered with a very thin skin. The ulcerated tit is the seat of an excessive granulation which is flattened over a small part of the skin surface, therefore of rather a pedunculated structure; this measures approximately one cm. at the base and two and one-quarter cm. across its surface. An area of induration extends from mid-axilla to mid-clavicular lines and up almost to the clavicle and below to the nipple line. The growth is apparently attached to the skin but moves freely, en masse, on its underlying structures. There is apparently no pain on palpation, the ulceration is painless, the entire mass is resistant in firmness but not hard. No pulsations are felt, but in places a distinct bruit is heard per stethoscope.

The supra- and infra-clavicular glands are somewhat enlarged on this side but not painful to palpation, and apparently *not* the seat of a very acute *inflammatory* process. The same region on the right side is negative, grossly.

The lungs are normal except over the outer infra-clavicular region, left side, in which area the resonance is impaired.

The left arm has a small mass in the upper third which is apparently composed of granulation tissue, approximately one and one-quarter cm. in diameter, flattened and closely simulating the tissue seen on the one ulcerated peak of the growth. Tissue is friable and bleeds slightly. The left axillary glands are enlarged. Supra-clavicle, left enlarged and one fairly discrete nodule is felt. Infra-clavicle, left enlarged, right axillary, supra- and infra-clavicular (epitrochelars), bilateral and inguinals (bilateral), are apparently normal.

On August 9, 1921, the growth was removed, together with the breast and underlying muscles, adopting the usual technic of a breast-cancer operation.

X-ray examination showed no signs of secondary bone involvement or any involvement of the lungs. The mediastinal shadow shows no enlargements.

The laboratory examination of the tumor removed. The specimens consisted of one large mass measuring twenty by six cm. at the base

and five cm. thick, and numerous small masses, for the most part lymphatic glands, of varying size. Main mass is moderately firm, the upper two-thirds covered with skin, the base is apparently covered on the under surface for the most part with a more or less formed membrane of connective tissue. Some muscle tissue is attached to the base edges. The upper surface of the mass has two projections, one apparently ulcerated and one a ruptured cystic formation. Color, blue-black. Cut surface is red and more or less smooth lobulations are lightly marked, numerous large vessels transverse in all directions. The surface directly under the dark projections is purple in color and run into the tumor in the shape of a cone or infarct (tip in). The small glands are hard, glistening and more or less friable (cut), with slightly increased resistance. Microscopic: Large round-cell sarcoma (alveolar). Glands show metastatic involvement.

The case made an uneventful recovery, gained weight and was apparently in good health on discharge. Was referred to the out-patient department for treatment by X-ray and the mixed toxins of Coley. This toxine treatment has been pursued with weekly injections to the present time. Examination within the last three days shows the left supra-clavicular space to be more full than previously noted. This change is recent, the space mentioned has always been somewhat full, but at present is more so, and two enlarged glands are easily palpated. Mother states that he is apparently normal.

PRIMARY AXILLARY LYMPHOSARCOMA IN A CHILD

Dr. J. Ralston Wells said, in studying this case of primary lymphosarcoma, alveolar in type, of the axillary lymphatics, he found that it was either of a relatively uncommon occurrence or else the reports in our literature for the last decade were not representative of its frequency.

A very brief résumé of this type of growth and its possible derivation, or better, theories as to its derivation, may not be amiss at this point.

Up to comparatively recent times all neoplasms were classed as cancer, but Virchow, approximately sixty years ago, first called attention to and designated sarcoma as a distinct group in this atypical growth of living tissue.[1]

Senn[2] defines sarcoma as "an atypical proliferation of connective-tissue cells from a matrix of fibroblasts of congenital or post-natal origin. . . . Connective tissue the sole origin of sarcoma . . . other tissues involved by extension. The intimate relations of the new blood-vessels with the parenchyma of the tumor is the characteristic feature of sarcoma. The more recent definition of Ewing[3] corresponds to that of Senn. "Sarcoma is a malignant tumor composed of cells of the connective-tissue type." This classification which in its basic points is clear, does not include a large number of tumors whose origin is questionable or whose structure is not typical and thus a border-line or transitional group of tumors are found. Ewing says that "Diseases such as angiosarcoma, lymphosarcoma and gliosarcoma are of such varied origin and character that some writers have urged the elimination of the term sarcoma. . . . The finer analysis of the origin and composition of many sar-

comas reveals a prominent participation of endothelium in many tumors of distinct mesoblastic characters. In such cases the characters of the tumor cells rather than their embryonal antecedents should determine the classification."

Lymphosarcoma is a true sarcoma, but it is also open to doubt in many border-line cases. Tracing this group, let us start from the purely benign lymphomata through the leukemias with their characteristic blood pictures, and pause a moment at Hodgkin's disease. This condition is relatively well defined, but its various gradations and locations very often lead us by logical sequence through Hodgkin's granuloma to Hodgkin's sarcoma and from this to true lymphosarcoma. Recorded cases of a seeming transition are found. Several striking examples by careful observers may be cited. A report by Welch,[4] in which he originally found Hodgkin's granuloma in a cervical lymph-gland, and some months later at autopsy, tumor masses resembling sarcoma were removed from the neck, dura, liver, etc.; another similar case by Karsner,[5] the following by Coley[6] in which a primary diagnosis of simple lymphoma was made (neck), seven months later a diagnosis of sarcoma. A true differentiation may be made between these border-line cases only when the relatively slight tendency to invade the surrounding tissues and the origin are taken into consideration.

The origin of sarcoma is one that has not been satisfactorily established, and it is not my purpose to enter into a lengthy discussion in this brief outline. True uncomplicated lymphosarcoma, localized or diffuse, is rapid in growth, and little, aside from the typical history and microscopic findings, are possible to determine theories of rests, inclusions, and undifferentiated cell groups. Occasionally a case of perhaps slower growth or a striking example of a particular type may be found, and these theories are not to be lightly laid aside, many appearing reasonable for specific cases; but in a large number of cases seen in the allied or borderline cases of Hodgkin's granuloma and sarcoma, and true lymphosarcoma, an irritant such as the tubercle bacillus or other like foreign agent seems to be a well-established origin.[7, 8] Whether a bacillus or other irritant starts the process, and this process then proceeds under its own momentum, or whether, as may be at times in the case of the tubercle bacillus, the bacillus breaks up into a granular formation and continues its activity in this state, is of no special consequence; the point is that a definite sarcoma, originating in lymphatic tissue, is found after passing through its "transitional" (possibly Hodgkin's granuloma) changes.[8] The exciting causes being bacillary or toxic irritation, repeated trauma, direct implantation, infection or transmission, with or without previous congenital inclusions. This may be more prevalent than has been realized. Many other explanations may be, and are possible, but at least this theory will be answerable for a definite number.[9, 10, 11, 12, 13]

Kundrat[14] separated definitely a type or class of lymphosarcoma from the general group of pseudo-leukæmia and leukæmia. This classification only holds for those that extend by definite lymph chains and channels. We know that true metastases does occur in lymphosarcoma other than by means of

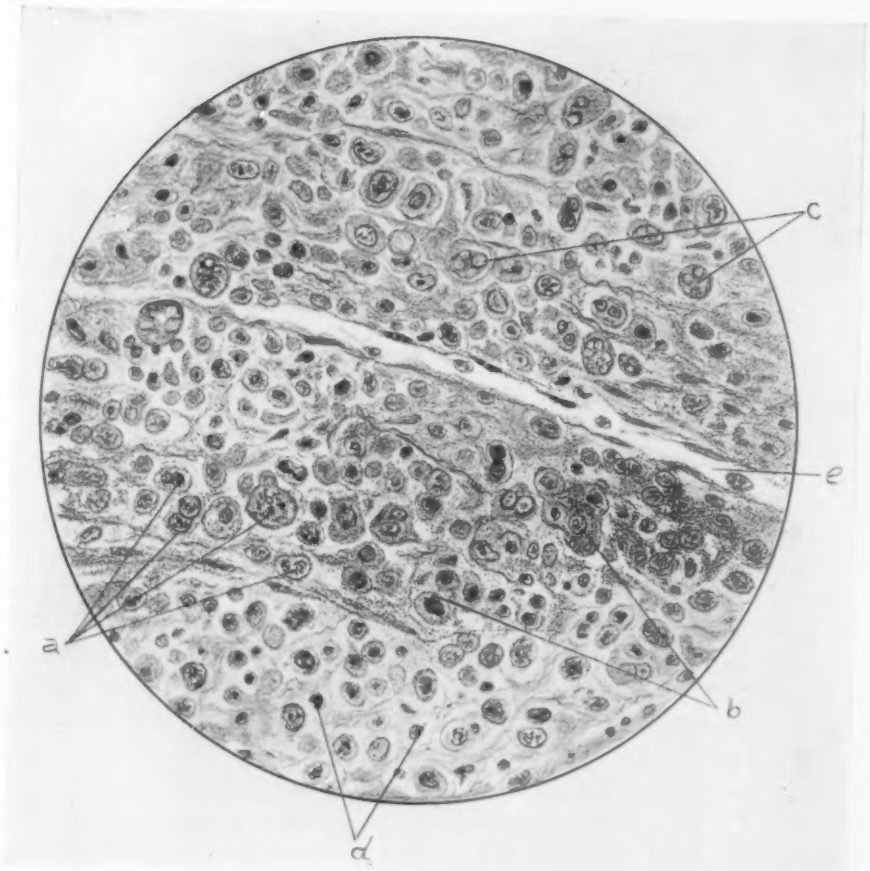

FIG. 1.—One microscopic field, not a composite drawing. a. Miototic figures and division. b. Alveolar groupings. c. Vacuolated neuculi d. Round cells. e. Capillary showing thin single celled wall.

the lymphatics, however Kundrat's group, he claims, is not to be confounded with the true neoplasms, although it is "one of the most malignant of diseases." Because this type of growth closely resembles pseudo-leukæmia, and that from the latter true lymphosarcoma may arise, we may infer a very close relationship to sarcoma, especially in view of the fact that we know of other changes of type.

Ewing states that there are two distinct forms of lymphosarcoma, namely: (1) Reticulum-cell sarcoma or large round-cell lymphosarcoma, and (2) malignant lymphosarcoma, (a) originating from the reticulum cell; . . . (b) from the lymphocyte. But "until the relation of the lymphocyte to the reticulum cell is fully established, the two conditions may be discussed together." Thus we see that the large class of lymphosarcoma is constantly changing as new findings are made; through the studies of many of our investigators, divisions and subdivisions are formed and as yet no classifications includes all established forms, even if the so-called borderline or transitional types were not taken into account.

As to the age incident, sarcoma is generally considered a disease of early life. This is true especially when contrasted to carcinoma, but it is also a disease of middle and advanced life. Therefore we must think of it as a disease of all periods of the life cycle.[15] It is interesting to note the age incidence of carcinoma and sarcoma; sarcoma occurs at an earlier age in the young than carcinoma, but both curves almost coincide from the ages twenty-eight to thirty-two up through age period forty-eight to fifty-two.

The most common seats of primary lymphosarcoma are (1) cervical (including the tonsils), (2) axillary, (3) inguinal, and (4) the retroperitoneal and mediastinal glands. The report of the sarcomas of the lymphatic glands (primary neoplasma of the lymphatic glands) by Coley [6] would lead one to think that the disease was more common than our investigations apparently show, but in this report we are hearing from a master surgeon in this particular field, and it is his exceptional opportunity to see a large number of this particular class of patients. His reports cover a period of twenty years up to 1915 and include cases of other surgeons' reports. The neck; neck and tonsils 103, other surgeons 22, total, 125; axilla 18, other surgeons 1; inguinal 17, other surgeons 2; retroperitoneal and mesenteric 10, other surgeons 12; mediastinal 1, other surgeons 0. An accompanying report of Hodgkin's comprises twenty-one cases. Investigation for a like number of years, 1902 to present, show in two large general hospitals; Hospital of the University of of Pennsylvania a total of twenty-three cases of true lymphosarcoma (neck 14, axilla 3, inguinal 1, all others 5); Philadelphia General Hospital a total of seven recorded.

For the privilege of operating upon this case and reporting it, he was indebted to Dr. W. E. Lee, on whose service in the Children's Hospital of Philadelphia the case was admitted and treated. At the time of this report the case is still under treatment.

BIBLIOGRAPHY

[1] Hunterr: Pediatrics, 1911, pp. 655–733.
[2] Senn: Path. and Treat. of Tumors.
[3] Ewing, J.: Neoplastic Diseases, 1919.
[4] Welch: New York Path. Soc., 1912–1913.
[5] Karsner: Arch. Int. Med., vol. vi, p. 175.
[6] Coley: Trans. of Am. Surg. Assoc., 1915.
[7] Flexner: J. Hopkins Hosp. Rep., vol. cxi, 1894.
[8] Ziegler, K.: Die Hodgk. Krank, Jena, 1911.
[9] Ricker: Arch. f. Chir., vol. l, p. 573.
[10] Brandts: Münch. med. Wochen., 1908, p. 735.
[11] Muller: (Dissert.) Ein Beitrog zur et des lymphosarcoma, 1894.
[12] Munk: Bruns Bert. z. kl. Chir., p. 60.
[13] Freudweiter: Deutch. Arch. f. kl. Med., vol. lxiv, 1899, p. 490.
[14] Kundrat: Wien kl. Woch., 1893, pp. 211–234.
[15] Weller: Arch. of Int. Med., 1915, vol. i, p. 518.

To Contributors and Subscribers:

All contributions for Publication, Books for Review, and Exchanges should be sent to the Editorial Office, 145 Gates Ave., Brooklyn, N. Y.

Remittances for Subscriptions and Advertising and all business communications should be addressed to the

ANNALS *of* SURGERY
227-231 S. 6th Street
Philadelphia, Penna